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DENTISTRY IN RELATION TO INTERNAL MEDICINE AND PREVENTIVE MEDICINE*

JOHN A. KOLMER, M.D., D.P.H., F.A.C.P., F.A.C.D.†

It has long been recognized that dental students and dentists require an adequate knowledge and experience in oral or dental medicine. This is true not only because various systemic conditions and diseases may be of dental origin but, even more importantly, because systemic conditions and diseases may produce oral manifestations. For various reasons, however, the education of dentists should be broadened to include many additional conditions and diseases in the domain of internal medicine because they are frequently in strategic position to first suspect or detect various systemic conditions and diseases without oral etiology or oral manifestations in patients unaware of their existence. Such knowledge and experience also enables dentists to cooperate more effectively with physicians in both diagnosis and treatment to the mutual advantage of both professions and the welfare of individuals. It also prepares them to better cooperate with physicians in the prevention of accidents incident to exodontia and oral surgery as well as in the prevention of various systemic conditions and diseases including some of the psychoneuroses. Furthermore, many phases of dental research bear an intimate relationship to internal medicine requiring in some instances cooperative efforts for the elucidation of many important problems.[1] But if this is true of dentistry it is also true that medical students and physicians require at least some instruction and experience in the principles and non-mechanistic phases of dentistry to enable them to better cooperate with dentists in the interests of the oral health of individuals and preventive dentistry.

EDUCATIONAL ASPECTS

Under the circumstances the course of instruction on internal medicine at Temple University School of Dentistry embraces 32 lectures at the School of Dentistry and 32 clinics at Temple University Hospital during the junior year, with 32 clinics at the hospital during the senior year, totalling 96 hours.[2] This is exclusive of sectional instruction in physical diagnosis with special reference to the cardiovascular system and student work in the medical clinic at the School of Dentistry. Special emphasis is placed upon those medical conditions and diseases of oral etiology and those characterized by actual or potential oral manifestations. At the clinics Dr. John Hamilton, of the faculty of the School of Dentistry, discusses the actual or potential oral aspects of each case presented. In this connection it is important that students should be prepared for such teaching by adequate instruction in the fundamental sciences during the freshmen and sophomore years with special reference to anatomy, physiology, biochemistry, pathology including certain aspects of clinical pathology, microbiology and pharmacology.[3]

At the present time some dental schools are using health questionnaires to be answered by patients as an adjunct to dento-medical diagnosis. Originally developed at Cornell Medical Center, the questionnaire now in use at Temple University School of Dentistry contains 82 questions, while that employed at the New York University College of Dentistry contains 205 questions for both sexes (34 of which are on oral history and symptoms), with 14 additional questions for women only, totalling 219 questions.[4] After being completed, the re-

† Professor of Medicine in the School of Medicine and the School of Dentistry, and Director of the Institute of Public Health and Preventive Medicine, Temple University, Philadelphia. Honorary Member, American Academy of Dental Medicine.

* Presented by invitation before U. S. Naval Dental School, April 3, 1953.

TABLE 1

POSSIBLE REASONS FOR INDIVIDUALS WITH PRIMARY FOCI OF INFECTION ESCAPING ILL HEALTH

1. Presence of local resistance and immunity: barriers of coagulated plasma, blockade of lymphatics and veins, fixation of bacteria and toxins, mobilization of leukocytes, phagocytosis, local antibody production, etc.
2. Presence of high natural nonspecific resistance to secondary infections due to good physical fitness, adequate capillary circulation, adequate production of ACTH and other hormones, etc.
3. Presence of high natural or acquired specific immunity to secondary infections due to phagocytosis and antibodies.
4. Absence of acquired allergic sensitization to bacteria or their soluble products in primary foci of infection.
5. Absence of production of substances promoting the spread of bacteria in primary foci and increasing capillary permeability (fibrinolysin, streptococcal proteinase, hyaluronidase).
6. Absence of factors favoring secondary localizations of bacteria during transient bacteremias (capillary occlusions, scarring, trauma, fatigue, etc.).

TABLE 2

POSSIBLE REASONS FOR INDIVIDUALS WITHOUT DETECTABLE PRIMARY FOCI OF INFECTION DEVELOPING CONDITIONS AND DISEASES COMMONLY ASCRIBED TO FOCAL INFECTION

1. Difficulties and failure to detect primary foci of infection of dental or other origin.
2. Possibility of transient bacteremias due to the lymphogenous or venous absorption of pathogenic organisms from presumably normal gastrointestinal, buccopharyngeal or other mucous membranes.
3. Presence of multiple primary foci of infection escaping detection.

TABLE 3

POSSIBLE REASONS FOR THERAPEUTIC FAILURE FOLLOWING THE REMOVAL, DRAINAGE OR DISINFECTION OF PRIMARY FOCI OF INFECTION

1. The presence of undetected and untreated multiple primary foci of infection.
2. Primary foci inaccessible to complete extirpation, drainage or disinfection.
3. Secondary diseases so chronic and well established as to be independent of primary foci of infection.
4. Secondary chronic infections and acquired bacterial sensitizations irreversible and beyond repair and recovery.

plies are checked and additional questions asked if necessary. Some dentists are also using such questionnaires in private practice with satisfactory results. Neurotic and psychoneurotic individuals, however, may answer "yes" to a bewilderingly large number of questions. Furthermore, the interpretation of "yes" answers to questions on medical conditions and diseases requires a substantial education and experience in internal medicine since it is frequently necessary to consider anywhere from three to ten or more different conditions or diseases from the standpoint of symptom diagnosis. Under these conditions the excellent book by Yater[5] can be highly recommended.

In this connection it is also advisable for dental interns in hospitals to make ward rounds with physicians at least once or twice a week.[6] This enables them not only to acquire more practical experience in internal medicine, but has also proven very helpful to medical interns and physicians in relation to dental diagnosis and treatment. Furthermore, it not only results in better cooperative medical and dental practice but demonstrates and teaches by actual example the great value of bringing the dental and medical professions into closer contact and consultation not only for the welfare of patients but in the interests of preventive medicine as well.

But, as previously stated, if such matters are of importance in dental education it is also true that there is a need for the adequate instruction of medical students and physicians in certain fundamental aspects of dentistry in relation to individual and public health, including preventive dentistry.[7] It is true that such instruction need not include operative dentistry and its mechanistic aspects, but devoted to the teaching of the important facts of dental structure and development and the recognition of dental defects and diseases. This would enable physicians not only to consult more effectively with dentists but place them in better position to give more adequate advice to patients in the prevention and treatment of dental diseases and

TABLE 4

EXAMPLES OF SYSTEMIC CONDITIONS AND DISEASES AFFECTING THE LIPS

Allergies to antibiotic compounds
Allergies to drugs and chemical agents
Angioneurotic edema
Granuloma inguinale
Histoplasmosis
Leprosy
Myxedema
Plummer-Vinson syndrome
Riboflavin deficiency
Tuberculosis (ulcers)
Syphilis (acquired)
Syphilis (congenital)

TABLE 5

EXAMPLES OF POSSIBLE CHANGES IN THE TEETH DUE TO SYSTEMIC DISEASES AND CONDITIONS

Alveolar atrophy due to diabetes mellitus or hyperthyroidism.
Caries due to prolonged febrile diseases.
Discoloration due to fluorine, copper, chromium, etc.
Discoloration due to endocrine disturbances, porphyrinuria or prolonged jaundice.
Enamel hypoplasia due to hypovitaminoses A and D.
Enamel hypoplasia and malocclusion due to congenital syphilis.
Increased sensitivity due to radiation therapy.
Neurility due to tabes dorsalis, multiple sclerosis or apoplexy.
Odontalgia due to tabes dorsalis.
Pain in upper teeth due to maxillary sinusitis.
Periodontal disease due to prolonged febrile diseases.
Pulpitis due to septicemia.
Root resorption due to hypothyroidism or Paget's disease.

defects than is now generally the case. Such instruction on dentistry in relation to medicine was introduced in Temple University School of Medicine in 1938 and has proven highly successful. At the present time it is largely given by my colleagues on the faculty of the School of Dentistry of Temple University, embracing a series of lectures on what dentists would like physicians to know about pedodontics, periodontia including gingivitis and gingivostomatitis, caries including endodontics, orthodontics, oral surgery and oral hygiene. In this connection it is also advisable for dentists to present papers or addresses before county, state, and national medical societies and for physicians to present papers or addresses before county, state and national dental societies more frequently than is now the case. Furthermore, it appears that at least one county society meeting per year could be profitably devoted to a combined meeting of dental and medical societies.

SYSTEMIC OR MEDICAL CONDITIONS AND DISEASES IN RELATION TO DENTISTRY

So many infectious diseases, intoxications, blood dyscrasias, avitaminoses, allergies, endocrinopathies, metabolic diseases, skin diseases and disorders of the nervous system may produce oral conditions and lesions that there can be no doubt but that the oral cavity reflects the state of systemic health more frequently than any other part of the body, aside from being one of the areas most vulnerable to endogenous and exogenous insults and irritants.[8, 9] Furthermore, some systemic conditions or diseases may originate in the oral cavity like those due to focal infection; gastrointestinal disorders, malnutrition and avitaminoses due to pain or edentulism; cancer; lung abscess due to dental foreign bodies; psychoneuroses due to harelip, cleft palate, etc. Of additional interest is the fact that certain systemic or medical conditions and diseases without oral manifestations are nevertheless in important relation to dentistry, with special reference to exodontia and oral surgery, like jaundice, paroxysmal tachycardia, hypertension, valvular and congenital heart disease, coronary arteriosclerosis, hyperthyroidism, diabetes mellitus, epilepsy, etc. Even if the dentist is too busy to inquire into the general health of his patients he should at least observe and advise medical consultations in such conditions that readily fall within his field of vision and examination, like the abnormal size and shape of the head, swellings of the face, increased pulsations in the neck, enlargement of the thyroid gland, cervical lymphadenopathies, etc.

TABLE 6

EXAMPLES OF SYSTEMIC CONDITIONS IN POSSIBLE RELATIONSHIP TO CARIES

Cretinism
Diabetes mellitus
Hyperthyroidism
Hypovitaminosis A
Pregnancy
Tuberculosis (chronic)

TABLE 7

EXAMPLES OF SYSTEMIC CONDITIONS AND DISEASES PRODUCING GINGIVITIS, PERIODONTITIS AND PERIODONTOSIS

Agranulocytosis
Allergies to drugs and chemical agents
Allergies to antibiotic compounds
Aplastic anemia
Blastomycosis
Capillary permeability (increased)
Diabetes mellitus
Dilantin therapy
Espundia
Gangosa
Gonadal dysfunction
Granuloma inguinale
Hyperthyroidism
Hypothyroidism
Leukemia
Menopause
Menstruation
Pinta
Pituitary dysfunction
Polycythemia vera
Pregnancy
Psychoneuroses
Puberty
Radiation therapy
Schüller-Christian disease
Sporotrichosis
Syphilis (early)
Tuberculosis (possibly)
Uremia
Vitamin B complex deficiency
Vitamin C deficiency
Yaws

During the past ten or fifteen years, however, no subject of dento-medical importance has created more discussion than focal infection in relation to health and disease. Undoubtedly many abuses, with special reference to the needless extractions of teeth and the removal of tonsils, have been perpetrated in its name. All of my experience as a bacteriologist, immunologist and internist during the past forty years, however, has left me with the conviction that focal infection of dental or other origin is a potential cause for various systemic conditions and diseases, as previously discussed in more detail.[10] It can be stated without fear of contradiction that focal infection is no longer a theory but a principle or concept firmly established in both medicine and dentistry. More individuals with primary foci of infection escape ill health than those developing various conditions and diseases. This is to be expected and could not be otherwise for reasons summarized in Table 1, with special reference to natural resistance, immunity, and acquired immunity. On the other hand, individuals may develop conditions or diseases commonly ascribed to focal infection without detectable primary foci of infection for reasons summarized in Table 2. Furthermore, it is to be expected that the removal, drainage or disinfection of primary foci of infection may fail therapeutically in many diseases of focal infection for reasons summarized in Table 3, although these procedures are frequently beneficial from the standpoint of improvement in general health.

As shown in Table 4, many systemic or medical conditions and diseases may affect the lips Likewise many may also produce changes in the teeth or their supporting structures, as summarized in Table 5. Indeed, while the exact etiology of caries is still unknown, or in dispute, there can be little or no doubt about the possibility of at least some systemic conditions having a relationship to etiology, as listed in Table 6. A large number of various systemic or medical conditions and diseases have even a more frequent and important relationship to the etiology of gingivitis, gingivostomatitis, periodontosis and periodontitis as shown in Table 7. Various pigmentations of the gingivae and of the buccal mucosa, listed in Table 8, may also be due to systemic diseases or intoxications. Possibly none of the systemic diseases,

TABLE 8

EXAMPLES OF POSSIBLE ORAL PIGMENTATION DUE TO SYSTEMIC CAUSES

Addison's disease
Acanthosis nigricans
Argyria
Auricism
Bismuthism
Chromiumism
Hemochromatosis
Mercurialism
Plumbism
Quinacrine therapy
Radiation therapy
Zincism

TABLE 9

EXAMPLES OF BLOOD DYSCRASIS WHICH MAY PRODUCE ORAL MANIFESTATIONS

Agranulocytosis
Aplastic anemia
Cooley's anemia
Hemophilia
Hypochromic anemias
Leukemia
Pernicious anemia
Plummer-Vinson syndrome
Polycythemia vera
Pseudo hemophilia
Purpura hemorrhagica
Telangiectasis
Sprue

however, produce as frequent or as much involvement of the gingivae and buccal mucosa, with or without secondary spiro-fusillar or bacterial infections, purpura and hemorrhage, than the blood dyscrasias listed in Table 9. Quite a number of conditions or diseases may produce glossitis, glossodynia or glossopyrosis as shown in Table 10. Many skin diseases may also produce oral manifestations (Table 11) even though the skin of the face may not be involved. There are many possible systemic causes for halitosis (Table 12) which should always engage the attention of dentists when purely local causes in the oral cavity are absent or eliminated. This is likewise true in dryness of the mouth (Table 13) and in the opposite

TABLE 10

EXAMPLES OF CONDITIONS POSSIBLY PRODUCING GLOSSITIS, GLOSSODYNIA OR GLOSSOPYROSIS

Gonadal dysfunction
Iron deficiency anemia
Leprosy
Lymphopathia venereum
Menopause
Myxedema
Nicotinic acid deficiency
Pellagra
Pernicious anemia
Plummer-Vinson syndrome
Psychoneurosis
Riboflavin deficiency
Sprue
Syphilis
Thiamine deficiency
Trichinosis
Trigeminal neuralgia
Tuberculosis

state of salivation with or without the dribbling of saliva (Table 14). As previously stated, dentists should always observe abnormal sizes and shapes of the head that may be due to any of the systemic or medical diseases shown in Table 15. The same is true of various swellings of the face due to inflammations or edemas produced by various infections or systemic diseases (Table 16). Pulsations of the carotid arteries or jugular veins, which are so frequently due to various and even serious systemic diseases (Table 17) should be brought to the attention of patients with the advice to seek medical assistance if they have not already done so. The same is true in the case of diffuse or nodular enlargements of the thyroid gland (Table 18) as, likewise, in the case of cervical lymphadenopathies detected by inspection and palpation (Table 19).

DENTISTRY IN RELATION TO PREVENTIVE MEDICINE

Primary prevention means the institution of measures for averting the occurrence of disease. Secondary prevention means the institution of measures for halting the progression of diseases from their early stages to more severe stages along with the prevention of accidents,

TABLE 11

EXAMPLES OF SKIN DISEASES WHICH MAY PRODUCE ORAL MANIFESTATIONS

Acrodynia
Dermatitis exfoliativa
Dermatitis herpetiformis
Epidermolysis bullosa
Erythema multiforme
Erythema nodosum
Herpes zoster
Impetigo contagiosa
Lichen planus
Lupus erythematosus
Lupus vulgaris
Molluscum contagiosum
Pemphigus
Pityriasis rosea
Sarcoid (Boeck's)
Scleroderma
Scleroma (rhinoscleroma)

TABLE 12

EXAMPLES OF POSSIBLE SYSTEMIC CAUSES OF HALITOSIS

Adenoids
Bromism
Bronchitectasis
Carcinoma of lung
Carcinoma of stomach
Diabetes mellitus
Dyspepsia
Iodism
Lung abscess
Plumbism
Rhinitis (atrophic)
Sinusitis (chronic)
Tonsillitis (chronic)
Uremia

TABLE 13

EXAMPLES OF SYSTEMIC CONDITIONS CAPABLE OF PRODUCING DRYNESS OF THE MOUTH

Atropine therapy
Brain tumors
Diabetes insipidus
Diabetes mellitus
Encephalitis
Gastritis (chronic)
Menopause
Nephritis (chr. glomerulo)
Psychoneurosis
Radiation therapy

TABLE 14

EXAMPLES OF SYSTEMIC CONDITIONS CAPABLE OF PRODUCING SALIVATION AND DRIBBLING

Erythema multiforme
Facial paralysis
Mercurialism
Neurosis
Paralysis agitans
Pemphigus
Trigeminal neuralgia

TABLE 15

EXAMPLES OF SYSTEMIC DISEASES PRODUCING ABNORMAL SIZE AND SHAPE OF THE HEAD

Achondroplasia
Acromegaly
Cerebral palsy
Cleidocranial dysostosis
Hydrocephalus (internal)
Leontiasis ossium
Mongolism
Myxedema
Osteitis deformans
Osteogenesis imperfecta
Richets
Syphilis (congenital)

TABLE 16

EXAMPLES OF SYSTEMIC DISEASES PRODUCING SWELLINGS OF THE FACE

Actinomycosis (cervicofacial)
Angioneurotic edema
Aneurysm (arch of aorta)
Anthrax
Glomerulonephritis
Ludwig's angina
Maxillary antritis
Mediastinal tumors
Mikulicz's disease
Mumps
Nephrosis
Trichinosis
Urticaria
Uveoparotitis

TABLE 17

EXAMPLES OF SYSTEMIC DISEASES PRODUCING INCREASED PULSATIONS IN THE NECK

(A)	Carotid arteries:
	Aneurysm (arch of aorta)
	Aortic regurgitation
	Cardiac hypertrophy
	Exopthalmic goiter
	Neurocirculatory asthenia
	Paroxysmal tachycardia
	Pernicious anemia
(B)	Jugular veins:
	Auricular fibrillation
	Congestive heart failure
	Heart block
	Mediastinal tumors
	Premature contractions
	Tricuspid stenosis

TABLE 18

DISEASES PRODUCING ENLAREGMENT OF THE THYROID GLAND

Adenomatous goiter
Carcinoma
Colloid goiter
Exopthalmic goiter
Sarcoma
Thyroiditis (acute and chronic)

complications, and sequelae as far as possible. Dentists cannot escape interest and responsibility in both phases in relation to the prevention of medical or systemic diseases along the lines summarized in Table 20. This is or should be in addition to preventive dentistry which so far has been devoted largely to oral hygiene and principles of dental prophylaxis, with special reference to the prevention of caries. That one disease may aggravate or complicate another is well known and designated as synergism; on the other hand, one disease or condition may ameliorate or improve another designated as antagonism.[11] As examples of synergism may be mentioned the transient bacteremias due to focal infections of dental origin in the etiology of subacute bacterial endocarditis in cases of valvular and certain types of congenital heart disease, with special reference to extractions of teeth; the dental defects of congenital syphilis in relation to caries; cleft palate in relation to sinusitis; diabetes mellitus in relation to pyogenic infections and pulmonary tuberculosis and obesity in relation to diabetes mellitus and hypertension. As examples of antagonism may be mentioned the improvement or subsidence of rheumatoid arthritis in pregnancy and jaundice as well as the improvement of rheumatoid arthritis, asthma and peptic ulcer in diabetes mellitus.

Chemoprophylaxis refers to the possible prevention of infections secondary to extractions of teeth and oral surgery by the administration of penicillin or other antibiotic compounds.[12] In relation to medicine this refers to the prevention of subacute bacterial endocarditis, due to infection with alpha or viridans streptococci, in cases of valvular and congenital heart disease; also to the prevention of infections in diabetes mellitus and the prevention of exacerbations or recurrences in iritis, iridocyclitis and other diseases due to focal infection. In relation to dentistry and oral surgery it also refers to the prevention of infections in plastic operations involving the mouth; surgical procedures associated with severe trauma; extractions of impacted third molar teeth; extractions in the presence of severe gingivitis; following accidental opening of the maxillary sinuses; following accidental injuries of the cheeks, tongue, lips or floor of the mouth; in compound fractures of the jaws and in fractures of the mandible involving impacted third molars. For these purposes procaine penicillin G in aqueous or oil suspension appears to be the antibiotic of choice. For adults it is advisable for the dentist, oral surgeon or physician to give 600,000 units of intramuscular injection at least 2 or 3 hours before extractions or other surgical operations, with a second dose 18-24 hours later and, possibly, a third dose 24 hours thereafter. The oral administration of penicillin is inadvisable although the oral administration of 500 mg. of aureomycin every 6 hours, around the clock, on the day before, the same day (not earlier than 3 hours before surgical measures) and the day after extractions or other surgical procedures, has been advised as being probably more effective than penicillin.[13]

TABLE 19

EXAMPLES OF SYSTEMIC DISEASES PRODUCING CERVICAL LYMPHADENOPATHY

Eczema
Hodgkin's disease
Infectious mononucleosis
Lymphogenous leukemia
Lymphopathiae venereum
Lymphosarcoma
Pediculosis capitis
Scrofula
Syphilis
Tonsillitis

TABLE 20

DENTISTRY IN RELATION TO PREVENTIVE MEDICINE

1. Dentists are frequently in strategic position to first detect or suspect the presence of systemic conditions or diseases in their early stages.
2. Prevention of conditions and diseases due to focal infection by maintenance of good oral hygiene, the prevention of caries and periodontitis, and good therapeutic dentistry.
3. Prevention of subacute bacterial endocarditis in individuals with valvular or congenital heart disease, infections in diabetes mellitus, exacerbations or recurrences of iritis, iridocyclitis, etc., due to focal infections, by the administration of penicillin in relation to exodontia.
4. Prevention of accidents in severe hypertension and coronary arteriosclerotic heart disease by alleviating fear and the proper choice of anesthesia in relation to exodontia.
5. Prevention of excessive bleeding in jaundice, hemophilia, pseudohemophilia, purpura, telangiectasis, etc., in relation to exodontia.
6. Prevention of gastrointestinal conditions and diseases by adequate prosthesis.
7. Prevention of psychoneuroses by adequate orthodontics.
8. Control of cancer by early detection and adequate treatment of oral precancerous lesions and incipient cancers.

SUMMARY

1. Dental students and dentists require not only adequate instruction and experience in oral or dental medicine but also in the broader aspects of internal medicine as well since they are frequently in strategic position to first suspect or detect various systemic conditions and diseases without oral etiology or manifestations in patients unaware of their existence.

2. This also enables them to cooperate more effectively with physicians in the prevention of conditions and diseases due to focal infection; in the prevention of accidents in severe hypertension, coronary arteriosclerosis and hyperthyroidism; in the prevention of excessive bleeding in jaundice and the blood dyscrasias; in the prevention of gastrointestinal disorders by adequate prosthesis; in the prevention of psychoneuroses by adequate orthodontics, and in the control of cancer by early detection and adequate treatment of oral precancerous lesions and incipient cancers.

3. Medical students and physicians also require at least some instruction and experience in the fundamental principles of the nonmechanistic phases of dentistry to enable them to better cooperate with dentists in the oral health of individuals and in preventive dentistry.

4. Undoubtedly the oral cavity reflects the state of systemic health more frequently than any other part of the body. Examples of systemic conditions and diseases affecting the lips, teeth, gingivae, periodontium and tongue are given in the text along with possible pigmentations, blood dyscrasias and skin diseases producing oral manifestations, halitosis, xerostomia and ptyalism, including examples of systemic diseases producing abnormal size and shape of the head, swellings of the face, increased pulsations of the neck, enlargement of the thyroid gland and cervical lymphadenopathies falling within the range of observation by dentists.

2101 Pine Street, Philadelphia 3, Pa.

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EFFECTIVENESS OF THERAPEUTIC DENTIFRICES

The need for greater accuracy in measuring the effectiveness of so-called therapeutic dentifrices, those that will actually prevent dental decay, was stressed recently at a conference of 25 of the nation's leading dental research scientists.

The conference was called by the American Dental Association's Councils on Research, Dental Health and Dental Therapeutics in an effort to establish minimum standards and procedures for clinical research in dentistry.

The scientists were sharply critical of incomplete or preliminary research reports which have been and are now being exploited by dentifrice manufacturers as the basis for sensational advertising claims for their products.

In summarizing the findings of the conference, Dr. Harold Hillenbrand, Association secretary, pointed out that the A.D.A. as yet knows of no satisfactory evidence that any dentifrice now on the market will actually prevent tooth decay.

Dr. Hillenbrand added that many of the misleading and distorted advertising claims now being made for dentifrices containing such substances as chlorophyllin derivatives, anti-enzyme chemicals and ammonia-containing compounds, were based on inconclusive or inaccurate measurements of their effectiveness.

The dental scientists participating in the conference agreed that experiments involving the control of dental decay should include the examination of all the teeth of individuals participating in the experiment rather than certain individual teeth or selected surfaces of the teeth.

Dr. Hillenbrand said that many of the claims for the so-called therapeutic dentifrices now being offered to the public were actually based on laboratory findings or on examinations of single teeth or a single tooth surface without regard to the condition of all teeth in the mouth.

NUTRITION AND THE CALCIFIED TISSUES*

JAMES H. SHAW, B.A., M.S., Ph.D.†

The last few years have been marked by an appreciable increase in the number of investigations concerned with the relation of nutrition to the development and maintenance of the bones and the teeth. In this area of research, as well as numerous others, it has become increasingly evident that the most important single component of our environment is the food that we eat. We can rise no higher physically than the base line that our day to day food intake permits. Unquestionably from individual to individual, the efficiency with which a given diet can be utilized varies widely depending upon the genetic constitution of the person and upon his or her current physical and emotional state of health. But irrevocably the most perfect constitution is bound to be penalized by inadequacies in food intake or by imbalances with which the body cannot cope indefinitely. There is an increasing volume of evidence in the nutrition literature to indicate that dietary abnormalities, which are sufficiently mild as to cause no immediate outward demonstration, often cause latent metabolic disorders or result in faulty structures which becomes manifest as disease entities later in life.

There are three logical divisions to a discussion on the relation of nutrition to the calcified structures (the bones, the enamel, the dentin and the cementum). Let us think of nutrition in relation to three particular intervals in the life history of these structures: first, the relation of nutrients to the development of the calcified tissues; second, the relation of nutrition to the maturation of those structures and third, the relation of nutrition to the maintenance of those tissues after they are fully developed and fully matured. The terms development, maturation and maintenance should not be used loosely, for it is true that, to a certain extent they represent eras which do merge and overlap one upon the other. Despite this overlapping, there is sufficient justification for a tentative segregation of these three time intervals in the life history of the calcified structures.

DEVELOPMENT

In the early days of the study of the relation of nutrition to the development of the calcified structures, it was very quickly evident that there were three particular deficiencies that resulted in abnormal formation of the bones and the teeth. The first of these was the group of defects in mineral metabolism associated with vitamin D deficiency and with aberrations in calcium and phosphorus intake and metabolism. The first extensive observations in this area were in the English laboratories of Sir Edward Mellanby (1918) who noted that experimental animals which were deficient in fat-soluble vitamins had great abnormalities in the formation of the bones. These syndromes came to be recognized as analogous to the rachitic process in the human being and were specifically related to the deficiency of vitamin D. Later, Lady May Mellanby (1918) observed that a deficiency of this fat-soluble vitamin in young puppies also had a profound effect on the developing enamel of the permanent teeth, and on the rate of eruption and on the position of the teeth in the jaw. When 8 weeks old puppies were maintained on a rachitogenic ration for 15 weeks, the enamel of the permanent teeth which was formed during that period was soft and brown, particularly at the necks of the teeth. The eruption of the permanent teeth was delayed and their arrangement in the jaw was very irregular. In the litter

† Assistant Professor of Dental Medicine, Harvard School of Dental Medicine, Boston, Mass.

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mates which received 10 gm. of cod liver oil per day as a supplement to the rachitogenic ration, the structure of the permanent teeth, their position and the rate of eruption were normal.

A deficiency of calcium in the dog in the presence of ample vitamin D (Mellanby 1923) resulted in some defects of the enamel and the dentin but these defects were not as severe as those produced by a deficiency of the fat-soluble vitamin. A deficiency of both calcium and vitamin D resulted in very badly formed enamel and dentin. If the cereal portion of the ration consisted of oatmeal or wheat germ, the tooth structures which developed during a period of deficiency were more defective than when white flour or rice were fed. Mellanby postulated a substance in the former cereals which inhibited calcification. However, the vitamin D in the cod liver oil was shown to overcome the adverse effect of the oatmeal or the wheat germ.

The profound effect of the diet on the female dog during pregnancy and lactation and upon the development of the deciduous and permanent teeth of the offspring was described by Mellanby (1928). When the diet of the female dog was low in vitamin D, the deciduous teeth of the puppies had typical defects in structure and their eruption was delayed. When the diet of the pregnant dog was supplemented with vitamin D, the offspring had normal deciduous teeth. If the puppies from a dog which had received ample vitamin D during pregnancy were placed on a rachitogenic diet at weaning, the deficiency produced milder changes in the enamel and dentin of the developing permanent teeth than it would have in puppies born to vitamin D deficient dogs.

The effect of vitamin A deficiency on the formative tissues of the incisor of the white rat has been described by Wolbach and Howe (1925[b], 1933). Atrophic changes in the ameloblasts were observed, followed by atrophy of the remainder of the enamel organ which finally consisted of the remnants of the epithelial papillae and squamous cells. The odontoblasts on the labial (enamel-covered) side retained their normal structure and function for relatively long periods of vitamin A deficiency. However, the odontoblasts on the lingual (cementum-covered) side became atrophic and depolarized simultaneously with the ameloblasts' changes. The dentin near the formative end on the lingual side became very thin and atypical, lacked the normal tubular arrangement and contained vascular inclusions. As a result of the active deposition of dentin on the labial surface of the incisor and the greatly reduced rate of dentin formation on the lingual surface, the dentin on the labial surface appeared extraordinarily thick when compared to the remainder of the tooth.

Schour, Hoffman and Smith (1941) confirmed and enlarged on Wolbach and Howe's observations. The primary effect of vitamin A deficiency was a failure of the odontogenic epithelium to undergo normal histo-differentiation, particularly on the lingual surface. Thus the normal organizing influence of the odontogenic epithelium to cause pulpal cells to differentiate into odontoblasts was lacking. The earliest response which could be recognized was a morphological and functional alteration of the lingual odontoblasts rather than a morphological change in the epithelium. Along with the failure of histo-differentiation, there was a continuation of the proliferation of the odontogenic epithelium which resulted in an invasion of the pulp by epithelial cords arising from the lingual odontogenic epithelium.

Boyle (1933) observed similar changes in the developing tissues of the incisor of a prematurely born infant which was believed to be vitamin A deficient.

Studies in the effect of vitamin C deficiency on the calcified structure were made by Jackson and Moore (1916) who observed not only the serious abnormalities of the periodontium but also noted pulp changes in the teeth of scorbutic guinea pigs and designated them as the processes of "necrosis." Höjer and Westin (1925) made a histo-pathological study of the jaws and teeth in scorbutic guinea-pigs. There was a gradual change and disappearance of the odontoblastic layer accompanied by an amorphous calcification of the predentin and the absence of Tomes' canals in this region. There

was commonly a dilation of the vessels of the pulp with occasional hemorrhage. Wolbach and Howe (1925a) noted that the ameloblasts continued to remain normal throughout prolonged deficiency processes but that the rate of dentin apposition was first slowed and then almost completely arrested.

Westin (1925, 1931) stated that the pathology in the pulp and odontoblastic layer of the teeth in scorbutic human beings were nearly identical with the pathologic changes observed in the incisor of the scorbutic guinea-pig. In the teeth of adults with scurvy, the dentin was found to be resorbed and porotic, particularly around Tome's canals. The little secondary dentin present was of the osteodentin type. The pulp was found to be atrophic and hyperemic. Degeneration of the odontoblasts, the formation of cysts and foci of denticle-like regions of calcification were observed.

These are three particularly clear cut deficiencies which cause specific definable abnormalities in the development of the teeth, and simultaneously, just as dramatic effects in the development of bones. Since the structure of the teeth is such that abnormal areas can never be reconstructed to erase these zones after the deficiency has been corrected, the tooth serves as a permanent kymograph to portray what occurred during its development. However, all deficiencies and abnormalities which occur during tooth development do not have sequelae that are revealed visualy by a structural examination of the teeth and their components. Therefore other criteria must be sought in the form of different chemical composition of the teeth, varying susceptibilities to tooth decay, etc.

The first major illustration of such a delayed criterion for demonstrating developmental influences of diet upon the teeth was presented by Sognnaes (1948). The results are depicted in Figure 1. When a natural ration, laboratory chow, was fed throughout the period of pregnancy and lactation and then to the offspring after they were weaned, the incidence of tooth decay was very low regardless of whether the

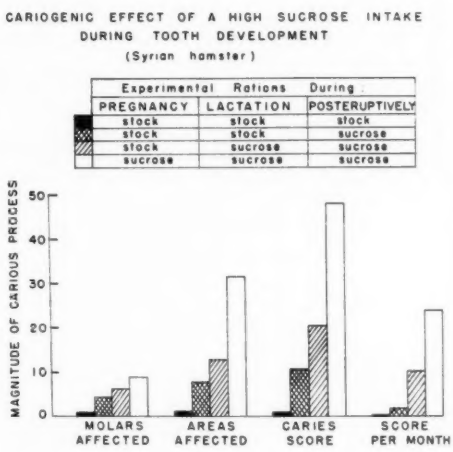


Figure 1

The caries incidence of groups of hamsters whose experimental feeding on the purified ration was commenced *before, during,* and *after* tooth developments, respectively; their molars decayed in a ratio of 20:10:2. ("Sucrose" diet refers to the purified ration, containing 67 per cent sucrose. "Stock" diet refers to the Purina laboratory chow.)

number of carious lesions or the caries score was used as the criterion. In other words, the natural diet through pregnancy and lactation when the molar teeth were developing and postnatally resulted in the initiation of practically no carious lesions. However, when the purified diet was used instead of the natural diet after weaning when the teeth were fully formed, there was an appreciable increase in the incidence of dental caries. This is an example of the difference between two diets on a post developmental or oral environmental basis. During tooth development, both groups of animals had been treated identically. After weaning, a natural diet produced essentially no caries; in contrast a purified diet with a high sucrose content post-developmentally resulted in an increase in tooth decay. However, there is one additional point of particular interest which is illustrated by the data in this chart. If the purified diet was fed to the mothers from the time the babies were born and fed throughout the lactation period, and then to

the babies thereafter, an appreciably higher incidence of caries and caries-score was observed. This second increase could not be explained on an oral-environmental basis. When the feeding of the purified diet was begun one stage earlier, i.e. before conception and fed through pregnancy and lactation and then to the offspring after weaning, still greater increases in caries incidence and caries-score were observed. Thus the teeth of the offspring of female hamsters fed the purified diet throughout pregnancy and lactation were 3 to 4 times as caries-susceptible as the teeth of the offspring of hamsters which were fed a natural diet throughout pregnancy and lactation. The same type of experiment with monkeys has been conducted with identical results (Shaw and Sognnaes, 1953). The teeth which were formed when the animals were in the jungles of India have not decayed even though they have been fed a purified ration with a sucrose content of 73 per cent for as long as 6 or 7 years, post-developmentally, but the teeth which developed in our laboratory while the animals were being fed the purified diet have had a relatively high caries-susceptibility.

It was of fundamental importance to explore why the natural diet promoted the formation of teeth which had a higher caries resistance than the teeth which were formed during the consumption of the purified diet. The mineral components of the natural diet were chosen as the initial area for investigation. As the source of these minerals, two hundred pounds of the laboratory chow were ashed at 1050° F. The resulting ash was used as the sole source of minerals in the purified diet in complete replacement of the usual reagent grade salt mixture. A similar experiment was conducted to that described previously, except that the natural diet was replaced by the purified diet which contained the mineral ash that had been prepared from the natural diet. (Sognnaes and Shaw, 1953). Under these circumstances the offspring of females fed the purified diet throughout the total experimental period from conception to sacrifice had the highest caries incidence (8.3 lesions per animal). When the ash diet was fed only during the lactation period and the purified diet during pregnancy and to the offspring post-developmentally, the caries incidence was reduced to 5.8 lesions per animal. When the ash diet was fed during pregnancy and lactation and the purified diet to the offspring post-developmentally the caries incidence was reduced to 3.5. When the ash diet was fed throughout the entire experiment from conception to the day of sacrifice, the caries incidence was reduced to 1.0. Thus the use of the ash of the natural diet as the mineral source resulted in almost as great a reduction in caries incidence as the natural diet itself.

Immediately the question arises, what is the fluoride effect in an experiment of this design? The natural diet would be expected to have some fluorides in it and it is true that the laboratory chow of common laboratory consumption contains in the neighborhood of 15 to 20 parts per million (p.p.m.) fluorides. This is a level just borderline, if not slightly below the minimum needed, to give any significant caries protection in the white rat, the hamster or the cotton rat. About 20 to 30 p.p.m. fluorides is needed in the diet to produce a comparable reduction in tooth decay in the white rat to what is known to occur in the human at the 1 p.p.m. level in the water supply. In this ash diet, the actual level of fluorine contributed by the ash to the diet was about 6 p.p.m., i.e., less than one-third of the level of fluoride that is known to be necessary to obtain a reduction in caries incidence. In order to confirm this postulate, an experiment was conducted with 6 p.p.m. of fluorine being incorporated into the diet as a supplement to the usual reagent grade salt mixture. (Sognnaes and Shaw, 1953). Absolutely no reduction difference in the caries index was obtained under controlled cricumstances. In other words, the fluoride content of the ash mixture was not the factor which resulted in the reduction in caries incidence when the ash mixture was the sole source of mineral nutrients.

This is a pertinent illustration of the relation of the diet during tooth development to the later caries-susceptibility of the teeth. It could be considered to be a "nutritional time bomb" where the predisposing dietary influence oc-

cured months before the disease entity was definable. If such conditions occur in human populations and there is every reason to believe that they do, the period of delay could be years rather than months because of the prolonged developmental period of the teeth.

An even more definitive experiment than these has been done by Sobel and co-workers at the Jewish Hospital of Brooklyn (Sobel and Hanok, 1948). Various dietary calcium and phosphorus ratios were imposed upon rodents during the time that their teeth were developing. For a number of years it has been known that moderate alterations in the calcium and phosphorus ratio of the diet caused definite alterations in blood. These investigators became interested in determining whether or not changes in the composition of the circulating blood would have an influence on the type of mineral structure which was being laid down in the bones and in the teeth of young, rapidly growing animals. This was an arduous experiment because of the detailed chemical methods required. Furthermore it was not a popular one by reason of the fact that the composition of the inorganic components of the bones and teeth was usually considered to be identical regardless of how or under what circumstances they were produced. The analytical data from these experiments demonstrated clearly that "bone salts" varied greatly in composition depending upon the pertaining metabolic circumstances. Not only did the calcium-phosphorus ratio of the diet alter the calcium-phosphorus ratio of the blood, but also caused appreciable alterations in the ratios of minerals which were being deposited in the bones and in the developing teeth. These changes were obtained with calcium and phosphorus alterations which were less severe than those required to produce a fully rachitic process. When a diet was fed which had a high calcium-phosphorus ratio, the mineral components in bones, enamel and dentin had a much higher carbonate-phosphate ratio than normal. When a diet was fed that had a low calcium-phosphorus ratio, the carbonate-phosphate ratio was decreased significantly. Since the hydroxyapatite with a high carbonate content has a much higher acid solubility than hydroxyapatite which has a low carbonate content, speculation as to the caries-susceptibility of the teeth of varying composition was a logical sequel. In a preliminary experiment, two groups of cotton rats, the one whose teeth had a high carbonate-phosphate ratio and the other whose teeth had a lower carbonate-phosphate ratio were fed on our typical purified cariogenic diet. (Sobel, Hanok and Shaw, 1953). Under these circumstances the cotton rats whose teeth had the high carbonate-phosphate ratio during development had a much higher incidence of carious lesions and a much higher caries score than the cotton rats whose teeth had the low carbonate-phosphate ratio. (Table 1). It is interesting to note that the teeth of the two groups of cotton rats were indistinguishable histologically, yet had these definite chemical differences and definite differences in caries-susceptibility.

TABLE 1

Effects of Developmental Diet on Caries Susceptibility

Diet	Number of cotton rats	Average number of carious lesions	Average extent of carious lesions
High Ca-low PO_4	55	11.2	26.0+
Low Ca-high PO_4	41	4.6	10.5+

MATURATION

The second phase of the relation of nutrition to the calcified tissues with which we should be concerned is maturation. The phase of maturation as I think of it begins when the teeth first begin to emerge into the oral cavity. At the beginning of that period the clincal crown has reached its full size, and the roots are relatively undeveloped. The period of maturation appears to be complete shortly after the tooth has fully erupted and is in functional position. There are a number of definite inferences in the older literature that once the tooth pierced the mucosa and began to erupt into the oral cavity,

the crown and its components were no longer subject to further systemic influences. Yet it has become increasingly evident that this is not so. Apparently there is a brief period after the emergence of the tooth into the oral cavity during which there is a considerable activity which is detectable by radioactive procedures. This activity is accompanied by an appreciable susceptibility to systemic influences. For the rodent molar this period probably lasts only a few days, while for monkey teeth it seems to last for several weeks. Definitive studies of this type have not been conducted for human teeth but presumably the period may be in the neighborhood of 3 or 4 months. It is not until the tooth is in full occlusion and full function that a final state of maturity appears to have been reached when there is a slowing down of these activities.

Of interest in the metabolism of the erupting tooth is the fact that it has a much higher ability to turn over radio-active phosphorus and presumably other ions also, than a tooth that has been in the oral cavity for a longer period of time. This is revealed by the radioactivity of the various teeth of monkeys which have had an injection of radio-active phosphorus 24 or 48 hours before being sacrificed. (Sognnaes and Shaw, 1952). An illustration of this is given in table 2. The average amount of radio-activity incorporated into the enamel of erupting teeth is 5 to 10 times greater than is incorporated into the enamel of a tooth which has been in the oral cavity for a long time. Similar but not as great differentials are noted for the dentin. This is not a simple surface phenomenon but exists in all levels of the enamel and dentin.

A second type of data which demonstrates again that there is a particular period in the life history of the tooth which can be characterized as the maturation period is shown in a recent experiment conducted in our laboratories. (Fanning, Sognnaes and Shaw, 1953). The experimental design was arranged to test the contribution of the salivary glands to the maturation of the teeth. Five groups of 12 rats each were desalivated at 21, 31, 41, 51, and 61 days of age, respectively. During this 40 day interval, all 60 animals were fed a completely non-cariogenic diet. On the 61st day of age, i.e. the day the 5th group was desalivated, all 60 rats were transferred to a cariogenic diet for an additional 120 days. Thus the only experimental difference between groups when the animals were sacrificed was the time at which desalivation was performed. The rats which were desalivated at 21 days of age had the highest incidence of dental caries; those which were desalivated at 61 days of age had the

TABLE 2

P^{32} distribution in premolars and second permanent molars at two developmental stages in intravenously injected rhesus monkeys

Tissue areas	Method of preparation	Developmental stage of teeth	No. of samples	Radioactivity* Range	Radioactivity* Average
External enamel	Grinding	In function	8	140- 442	240
		Erupting	5	659-1,860	1,190
Internal enamel	Grinding	In function	4	37- 147	89
		Erupting	4	236-1,110	346
Whole enamel	Bromoform separation	In function	8	52- 144	102
		Erupting	17	145-2,760	1,390
Whole dentin	Bromoform separation	In function	7	372-1,540	798
		Erupting	15	598-3,030	1,650

* Values expressed in number of counts per minute per milligram of tissue.

lowest caries incidence; the groups desalivated at intermediate ages had progressively less caries as the age at desalivation increased. In other words, the presence of saliva in the oral cavity appeared to contribute to the maturation of the teeth in such a way as to cause them to attain a great caries resistance when there was a salivary contact throughout the full maturation period.

A further experiment which may shed some light on the process of maturaton is one in which an experimental group of rats was fed the purified ration entirely by stomach tube while the littermate controls were allowed to eat the same diet in the normal fashion. (Kite, Shaw and Sognnaes, 1950). The molar teeth of mature tube-fed rats were completely impervious to histological staining procedures; in this regard, the teeth of this group were histologically similar to the teeth of rats of the same age which had been fed natural diets. However, in contrast, the molar teeth of the rats which were allowed to eat the purified ration were stained to some extent by the same histological procedures; thus the teeth of these mature rats histologically resembled the state of immaturity seen in recently erupted teeth. It would seem from these data that the presence of the purified ration in the oral environment around the newly erupted teeth of these rats had retarded or arrested the normal process of maturation.

These three demonstrations that the period after teeth erupt is one of potential importance in the overall life history of the teeth do little to help us understand what is going on at that time. There is a real need for definitive experiments concerning what is taking place during maturation, what is the full importance of this stage, how the teeth can be protected during this era of apparently high disease susceptibility and how the process of maturation can be augmented as a means to increase the disease resistance of the teeth.

MAINTENANCE

Is there any possible way in which nutrition through systemic influences can play a significant role in the maintenance of teeth? This is a question which has been asked repeatedly in the past decades with the forthcoming answers varying through all parts of the spectrum. As far as bones are concerned, we know that there is a normal reworking of the Haversian canal systems. Furthermore, where calcium-phosphorus vitamin D metabolism is

TABLE 3

P^{32} distribution in fully erupted first permanent molars of 11 rhesis monkeys after intravenous administration of 6 to 18 millicuries of P^{32}

Tissue areas	Method of preparation	No. of samples	Radioactivity* Range	Average
Enamel—external layer	Grinding	9	87- 258	169
Enamel—second layer	Grinding	4	34- 191	63
Enamel—third layer	Grinding	6	15- 61	26
Enamel—fourth layer	Grinding	3	7- 20	14
Dentoenamel junction	Grinding	6	16- 68	42
Dentin—external layer	Grinding	7	29- 119	56
Dentin—second layer	Grinding	9	145- 285	208
Dentin—third layer	Grinding	10	238- 540	407
Predentin	Grinding	6	1,080-2,540	1,390

* Values expressed in number of counts per minute per milligram of air-dried samples.

deranged, osteomalacia occurs in the adult. In others words, there can be a leaching out of the already-deposited calcium salts in the long bones which will cause an adult form of rickets. Is there any comparable relationship to the mineral salts in the enamel and dentin and the maintenance of the teeth? On the basis of chemical analyses, there is no evidence that a comparable process occurs in the teeth. However, this does not mean that the components of the teeth have no capability for metabolic functions of a less readily demonstrable type.

The use of radioisotopes to explore the possible metabolic processes in the enamel and dentin has provided many interesting facts (Sognnaes and Shaw, 1952). The most intensively used of the radioisotopes has been radiophosphorus which serves as a tracer to indicate to what extent phosphorus metabolism is taking place. In studies with the rhesus monkey, the enamel and dentin of fully erupted permanent molars were found to take up appreciable amounts of radiophosphorus after intravenous administration of this isotope. From the data in table 3, it can be seen that there is no part of the enamel and dentin into which the radiophosphorus has not been introduced. Indeed two striking gradients were observed, one decreasing from the external to the internal enamel and the other increasing from the external dentin to the internal dentin. The average of all values in the enamel and dentin, respectively, indicated that the average turnover in the dentin was approximately five times that in the enamel. One of the most important observations was that the elevated activity of the surface enamel could be eliminated by isolation of the teeth from any contact with the radioactive saliva produced by the monkey after isotope administration. Thus the majority of the radiophosphorus which was incorporated into the enamel during these trials had originated in the saliva. Likewise, the higher levels of radiophosphorus observed in the dentin of isotope-injected monkeys could be abolished by removal of the pulpal blood supply. It is important to note in passing that this incorporation of phosphorus into all regions of the enamel and dentin was not merely a passive process but in order to occur at its maximum rate, the tooth has to be vital. This was demonstrated in an experiment where the isotope uptake of extracted teeth was compared with that of vital teeth in situ in the monkey jaw. Each tooth was bathed by a comparable radioactive saliva mixture in a plastic cup sealed around its neck. In all cases, the teeth in situ in the jaw had incorporated 4 to 9 times as much of the radioisotope into their mineral components. Thus from these various trials, it is readily evident that functioning mature teeth have metabolic interrelationships to both the saliva bathing the external surface of the enamel and to the blood vascular network within the pulp. Between these two pathways, no area in the calcified tissues of the teeth is inaccessible to mineral nutrients, albeit the potential rate of interchange is a great deal slower than is known to exist in the bones.

Another series of studies which indicate that metabolic potentialities exist in the teeth after they are fully formed has been conducted in cooperation with Drs. Haldi and Wynn of Emory University. The Emory workers had always reported a much lower caries-susceptibility for the rats on experiment in their laboratories than we had been reporting from the Harvard laboratories. In discussions, it appeared that in practically every regard our experimental circumstances were the same. Hence we began to pool experimental resources to determine what there was about our regimens that caused the difference. The first procedure was to compare on a post-developmental basis the effect of the Harvard and the Emory diets when run parallel to each other in a single laboratory with a single strain of rats. (Wynn, Haldi, Shaw and Sognnaes, 1953). These two diets are closely alike in their carbohydrate, protein and fat composition, but differ slightly with respect to their mineral components and yet not in ways which on superficial examination would be expected to be of importance. It is noteworthy that in these post-developmental studies the animals were weaned prior to the experimental shift in diet. At the end of the experimental period the group of rats fed the Harvard diet had a much greater caries in-

cidence than the rats fed the Emory diet. (Table 4). The only logical interpretation that can be suggested for this post-developmental influence is that there has been some nutritional factor in operation. Possibly this influence has not operated through the teeth themselves, but perchance through the saliva or some other pathway. What factor or factors of the diet was involved is unknown at present. However, this is an example of a post-developmental effect where the component of the oral environment attributable to the diet seems to be identical by reason of the comparable fat, carbohydrate and protein concentratiron.

An investigation of similar import has been described in hamsters by Nizel and Harris (1951). Hamsters which were fed a diet composed of corn grown in New England and whole milk powder prepared from milk produced in New England had a high dental caries incidence. Littermate hamsters fed a diet composed of corn and whole milk powder produced in Texas had a significantly lower dental caries incidence. On the basis of chemical analyses, these diets were highly similar. Yet on a post-developmental basis, they had produced extremely different results with respect to tooth decay production in the hamsters.

An experiment which was even more striking has been published recently by McClure and Folk (1953) at the National Institutes of Health. When skim milk powders were heated briefly under relatively mild conditions, appreciable increases in their caries-producing properties for rats were observed. (Figure 2). An interesting thing about this post-developmental experimentals that once again the carbohydrate, fat and protein percentages were identical before and after the heating process for each of the three skim milk powders. The diets (A, C and E) which contained an unheated skim milk powder caused a relatively low dental caries incidence; on the other hand the diets (B, D and F) which contained the same three skim milk powders after a mild heat treatment caused a much higher incidence of dental caries. This effect does not appear to be explicable on the basis of the direct effect that these diets would have upon the oral environment of the teeth.

A particularly valuable illustration of the strong metabolic relationships to caries-susceptibility is the wide variation between strains of laboratory animals with respect to dental caries incidence when representatives of the strains are maintained under identical experimental circumstances. The caries-resistant and caries-susceptible strains of the Michigan State College laboratories are a good example of this hereditary difference. Stewart. Hoppert and Hunt (1953) noted that on a natural diet representatives of their caries-resistant strain did not develop grossly demonstrable carious lesions until an average of 372 days. Many of

TABLE 4

Dental Caries in albino rats and cotton rats fed the Emory and Harvard diets[1]

Diet	Number of Animals	Average Number of Carious Molars	Average Number of Carious Lesions	Average Caries Score
ALBINO RATS				
Emory	24	5.1 (± 0.6)	7.3 (± 1.0)	24.9 + (± 3.9)
Harvard	24	10.2 (± 1.4)	21.8 (± 1.1)	83.8 + (± 5.4)
COTTON RATS				
Emory	17	3.6 (± 0.4)	7.0 (± 0.8)	15.4 + (± 2.1)
Harvard	20	6.0 (± 0.7)	13.4 (± 1.7)	41.9 + (± 7.6)
Stock diet[2]	17	1.0 (± 0.6)	2.0 (± 0.1)	3.8 + (± 2.5)

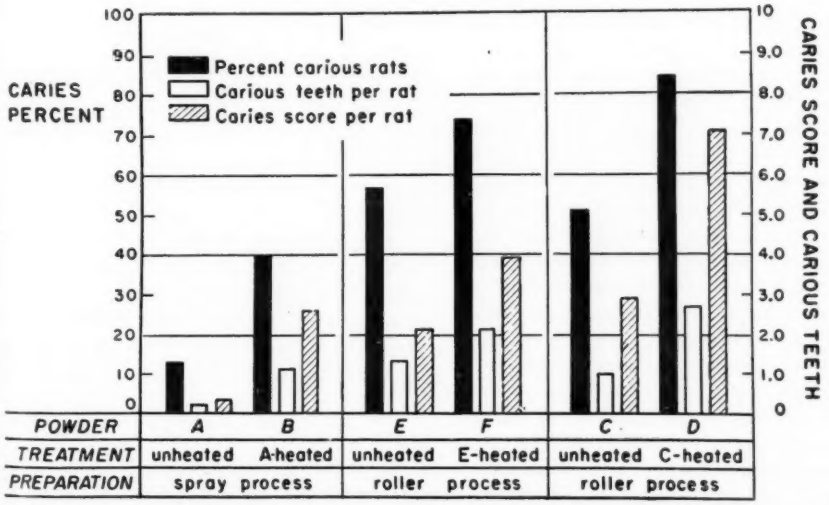
[1] The figures in brackets are the standard errors of the means (S.E.M.).
[2] Purina Laboratory Chow.

the caries-resistant rats still were caries-free when the test period was terminated at 500 days. In contrast, the average time of appearance of carious lesions was 58 days for the representatives of the caries- susceptible strain. Aside from the different genetic backgrounds of the two strains, all these representatives had been treated identically with respect to the diet to the mothers, during pregnancy and lactation, the housing conditions, the drinking water, the post-natal diet, etc.

Similar differences between strains have been noted in the Harvard laboratories. One is a modification of the strain of rats that has been used at the Navy Dental Research Institute for several years. When representatives of this strain are maintained on even moderately cariogenic diets for 3 to 4 months, the teeth have been destroyed to the point where little more than the roots is left. The other strain of rats was developed by a breeding program in our laboratory during the last 8 years. Representatives of this strain have teeth which last almost indefinitely without any destruction by reason of tooth decay. As far as animals from these strains are concerned, they appear to be identical in numerous respects: growth rate, adult weight, reproductive ability, hemoglobin concentration, and red blood cell count. They appear to need the same nutrients in similar amounts; although we have not studied the latter relationships with the thoroughness that is required for an exhaustive knowledge of this subject. However, regardless of how many ways in which the representative of these strains resemble each other, it is obvious that through some unknown systemic pathways which are products of their hereditary background, they differ widely in their ability to compete against the oral environment produced by cariogenic dietary regimens. In this particular field of dental research there is a tremendous opportunity to find out why the teeth of these animals have such vastly different abilities to withstand the

Figure 2

EXPERIMENTAL RAT CARIES



Incidence and severity of dental caries in white rats fed diets containing heated and unheated dry skim milk powders.

oral environment to which they are subjected. Is it a matter of tooth composition or of tooth form and position? Is their saliva of strikingly different composition? Do they have different abilities to utilize the nutrients provided to them? The answers to these questions and to numerous others which could be asked with equal justification are unknown. Somewhere in the constitutional differences between these two strains lies a very important answer to one of the world's most prevalent diseases.

SUMMARY

There are three axioms pertaining the relationship of nutrition to the calcified structures. The first is that the bones and the teeth continually are in a dynamic state throughout their life histories. In teeth this process is sufficiently slow that on the basis of the best microchemical analytical procedures it has never been possible to demonstrate a difference in the inorganic components of the enamel and dentin as a result of the most drastic post-developmental alterations in the diet. A slow turnover of the minerals in enamel and dentin can be clearly demonstrated when radioisotope tracers are used. The rate is greater in vital than in extracted teeth and in immature than in mature teeth.

Secondly, the mineral components of bone, enamel and dentin are deposited in different proportions which are dependent upon the available nutrients, the hormonal influences, the organic disease components of the individual at the time these tissues were being formed. There evidently is no such phenomenon as a constant proportion of mineral components being deposited in these tissues irrespective of the systemic circumstances current at the time that these salts are deposited. Instead the proportions of inorganic components vary with the prevailing conditions.

And third, it is axiomatic with investigators today that there are strong systemic differences between strains of animals. For this reason, no satisfactory experiment on dental caries can be conducted with laboratory animals of unknown strain, where dietary background is unknown or without littermate controls.

188 Longwood Ave., Boston 15, Mass.

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THE QUEST FOR EARLY ROENTGENOGRAPHIC SIGNS OF RETROGRESSIVE ALVEOLAR BONE CHANGES IN CHILDREN: AN INITIAL STUDY AND EVALUATION*

GEORGE J. WHINSTON, D.D.S.†

INTRODUCTION

A unique type of advanced alveolar bone resorption has been referred to on various occasions as periodontosis, precocious advanced alveolar atrophy, and diffuse alveolar atrophy. Periodontosis, as defined by the 1949 Nomenclature Committee of the American Academy of Periodontology,[1] is a general term used to include a degenerative non-inflammatory destruction of the periodontium, originating in one or more of the periodontal structures. It is characterized by migration and loosening of the teeth in the presence or absence of secondary epithelial proliferation and pocket formation or secondary gingival disease. Precocious advanced alveolar atrophy is a descriptive term used by Miller, Seidler, and Wolf[2] for those cases of advanced periodontal disease in young patients in which local factors were not severe enough to cause the existing tissue destruction. It is their contention that some systemic defect was mainly responsible for the changes which were observed. Gottlieb,[3] as far back as 1925, presented his views on diffuse alveolar atrophy. He considered this type of bone destruction to be intimately associated with a disturbance in cementum formation, affecting the collagen fibers of the periodontal membrane and ultimately the alveolar bone.

The etiology of this category of alveolar bone disturbances has been the subject of considerable investigation through the years. This study was undertaken in order to determine whether early signs of advanced alveolar bone resorption may be detected in the permanent dentition of apparently healthy children. An attempt will be made to correlate these findings with other factors in the background of the cases studied. Such an investigation may very well increase our knowledge of the effects on alveolar bone development in this early age group. The findings may serve both as a stimulus and as an indicator for future studies of the nature of the involved mechanisms. In this way we may be aided in our basic approach to the problem.

REVIEW OF THE LITERATURE

Numerous studies have been conducted in laboratories to determine a condition or group of conditions which would cause such a characteristic destruction of the alveolar bone. At the same time, there have also been investigations to determine clinically the etiology, incidence, and onset of this condition. A survey of the literature will clearly illustrate the diversity and abundance of such studies.

Boyle[4] has demonstrated through extensive laboratory studies the effects on guinea pigs of diets deficient in vitamin C. A marked rarefaction of the alveolar bone and a widening of the periodontal membrane space occurs. He has stated[5] that the gross, roentgenographic, and histologic changes in the periodontal tissues of the guinea pig maintained on such a diet are typical of scurvy and reproduce the diagnostic features recognized as characteristic of human diffuse alveolar atrophy.

Boenheim[6] observed that the anterior lobe of the pituitary, the thyroid, and the ovaries are associated with periodontosis. To a lesser degree, the same is true of hyperparathyroidism and disorders of the adrenals. Orban and Weinmann,[7] in 1942, pointed out that intrinsic factors must be considered primarily as a cause

† Assistant, Department of Periodontia, New York University College of Dentistry.

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for the initiation of the pathological changes in the alveolar bone. These factors may be the dysfunction of different endocrine glands, toxins from various sources, metabolic disturbances, and dietary deficiencies.

Glickman, in a series of studies, has attempted to demonstrate the significance of systemic influences on the periodontium. Experimentation in the laboratory by Glickman brought forth information concerning the effect of such factors as diabetes, vitamin C deficiency, protein deprivation, and cortisone on the periodontal structures. Using rats, Glickman[8] experimentally produced diabetes by injecting a drug, alloxan. After studying the tissues of these animals, he made the following observations regarding human diabetes: (1) Diabetes is not responsible for specific gingival changes or for the onset of gingival disease. (2) The progress of periodontoclasia in diabetics in whom the alveolar bone is affected by generalized osteoporotic changes may expectedly be more rapid than that which would occur in the absence of diabetes. (3) The existence of a basically unaltered condition throughout the alveolar bone in a large percentage of cases discourages the assumption that whenever periodontoclasia occurs in diabetes its occurrence is due to the disease. (4) Notable changes in the periodontal membrane or cementum are not associated with diabetes. Using guinea pigs in another experiment,[9] Glickman described the microscopic changes occurring from vitamin C deficiency. In the absence of gingival inflammation or pocket formation, vitamin C is responsible for destructive changes in the periodontal membrane and alveolar bone. The condition produced may be described as generalized periodontoclasia or diffuse alveolar atrophy. The effect of protein deprivation on the albino rat, as shown by Glickman and Chawla,[10] abnormally affects the gingiva, the periodontal membrane, the alveolar bone, and the cementum. The loss of bone is not due to destructive changes, but rather to reduced formative processes. The most recent study reported by Glickman and his associates[11] reveals the effects of cortisone injected into white mice. The following changes were observed microscopically: osteoporosis of the alveolar bone characterized by a marked reduction in osteoblastic activity, capillary dilatation and engorgement with hemorrhage in the periodontal membrane and gingival connective tissue, and reduction in the number and degeneration of the collagen fibers of the periodontal membrane. Gingival inflammation was more marked in the animal receiving cortisone.

Karshan and his associates have conducted several studies which have revealed certain other aspects of this condition. In their earliest studies[12] of the blood chemistry of patients with advanced periodontoclasia, significant differences between the means of the control and periodontoclasia groups were found for serum phosphatase, erythrocyte sedimentation rate, total serum protein, serum globulin, leukocyte count, basal metabolism, serum calcium, and sugar. In an effort to determine the ovarian function in patients with periodontosis, a series of studies was later undertaken.[13] These studies were based on the estrogen content of the urine on the seventh day of the menstural cycle, by serial vaginal smears on alternate days during a complete cycle, and basal body temperature recordings. Of 20 cases studied, the estrogen output in the urine samples collected was within the accepted normal limits. However, on the basis of the smear picture and temperature recordings, half of the patients studied showed some impairment in ovarian function. In a more recent study made of 77 periodontosis cases,[14] physical examination and case histories were negative in 17 of them. Several apparently unrelated conditions were revealed in the remainder of the group. It was also found in this particular study that the plasma vitamin C was low in a large percentage of periodontosis cases in spite of an adequate intake of this vitamin. This could indicate that there was some interference in the utilization of the vitamin. Although a difference was found between the control and the periodontosis group for the ketosteroid content of the urine, it is as yet uncertain whether adrenocortical dysfunction is related to periodontosis. (The determination of the 17-ketosteroid output is essentially an index of

adrenocortical and gonadal activity). Since deviations from the normal were obtained for several apparently unrelated blood constituents, it does not seem as though a common systemic condition acts as a predisposing or modifying factor in these conditions. In an effort to determine a relation between diet and periodontal disease, a record was made of the food consumed during a one week period for 22 control and 24 periodontosis cases.[15] It was found that the caloric intake was low in 45% of the control group and 48% of the periodontosis group. Similar results were found for protein, calcium, phosphorus, iron, thiamin, riboflavin, and niacin. In general, there was no difference between the two groups. There was evidence in this study, however, of excess carbohydrate intake in the periodontosis group.

Some interest has been shown concerning emotional and psychosomatic relationships in advanced alveolar bone resorption. In a study of 10 cases of periodontosis, it was observed by Campbell[16] that patients with this condition have moods of sullen resentment which are often cleverly disguised. There is a tendency for these individuals to be overprotected, pampered, cautious, conventional, and orderly. As safety devices to maintain their security, these patients may restrain their aggressiveness and consequently suffer from guilt feelings and depression. The author further states that the pathogenetic influence of such emotional disturbances may be of two types, direct and immediate, or indirect. The direct influences act through rapid and severe alteration of the physico-chemical equilibrium (homeostasis) by way of the autonomic nervous system. The indirect type acts through the delayed mechanisms of faulty oral sanitation and nutritional habits which are the reflection of character trends such as infantile habits of sucking, grinding, and candy eating. In contrast, Moulton, Ewen, and Thieman[17] in a more recent study referred to overt habits as the direct routes while the physiologic mechanisms utilizing the autonomic nervous system were considered the more indirect routes. Included in the total number of cases studied were 10 cases of periodontosis, 1 male and 9 female. These patients, unlike the other groups which included acute necrotizing ulcerative gingivitis and chronic periodontitis, had less evidence of neurotic tendencies and destructive oral habits. These findings suggested that degenerative oral processes are less associated with emotional conflict than are the inflammatory processes involving the soft tissues.

Miller[18,19] has described three types of precocious advanced alveolar bone destruction:

Type 1—Early—probably initiated between 5 and 7 years of age. The incisors and first molars are involved while the other alveolar bone is essentially normal.

Type II—Late—probably initiated between 11 and 13 years of age. The canines, premolars, and second molars are involved while the other alveolar bone is essentially normal and not involved.

Type III—Combined—early involvement of all teeth due to continuing destructive influences.

What results is a condition of osteogenesis imperfecta. It is very likely that the bone loss occurring in the early age groups listed above is due to (1) a rapid growth period where metabolic changes are occurring and ultimately creating a disturbance in bone formation and (2) emotional disturbances resulting from the child's separation from his mother when starting school and his taking on added responsibilities. This emotional upheaval may result in bodily stresses leading to osteoporosis and to such habits as clenching and bruxism at the time the particular teeth are erupting. This work was further elaborated upon by Miller and Seidler[20] in a study of patients between the ages of 13 and 30. It was noted that there was a greater amount of bone loss in the incisor and first molar regions of both jaws. It was suggested that the bone destruction may be of greater severity in these areas because these teeth are the only permanent teeth in the mouth at the age of 7 and consequently may receive an excessive stress at that time. Considering all the teeth to have been weakened equally by a systemic background, then the teeth which have shown the greatest breakdown

are naturally the ones which have received the greatest total stress. At a certain point, the alveolar bone suddenly begins to disappear. Consideration must be given to metabolic and nutritional disorders existing during the eruption of the involved teeth which may have weakened their alveolar processes. In a more extensive investigation by Seidler, Miller, and Wolf,[2] thirty-five cases of precocious advanced alveolar bone destruction ranging in age from 14 to 40 were examined to determine a systemic relation. Females constituted the greatest number in this age group. Roentgenograms of the skeletal bones revealed nothing unusual. Eighty per cent of the patients consumed excessive amounts of-carbohydrates. The onset of the disease appeared to be around puberty (13 years old) and in males slightly later. The outstanding laboratory deviations were: phosphatase activity lowered, specific dynamic action of protein lowered, cholesterol values increased, basal metabolism lowered, and a flattened glucose tolerance curve.

Burket[21] has recently presented an extensive survey of the relationship of systemic disturbances to periodontal disease. He emphasizes that the chronic mild nutritional deficiencies still represent the most important group of diseases which may predispose to periodontal disease. Concerning the relationship of uncontrolled diabetes to periodontal disease, Burket discusses three indirect effects of the disease on the supporting structures. (1) Since diabetic patients have a decreased ability to combat infection, the role of non-specific bacterial factors may be accentuated in the causation of periodontal disease. (2) In diabetes there is a depressed activity of vitamin C and increased requirement of the vitamin B complex group. These conditions may have a detrimental effect on the periodontium. There is a similarity of oral symptoms between uncontrolled diabetics and the vitamin B complex deficiency. (3) Due to a resulting reduced alkaline reserve in the uncontrolled diabetic, there may be an increased tendency for calculus to be deposited. This will contribute locally to the onset of periodontal disease.

It is readily seen from this review that there has been considerable research to determine what relationship advanced alveolar bone destruction may have with the various blood constituents, nutritional deficiencies, emotional disturbances, and systemic abnormalities. The studies on any one phase have been manifold. Some of these have confirmed one another, while others have presented diametrically opposite views. This all the more emphasizes that no definite answer to the problem has been obtained. Additional investigation is still necessary.

PURPOSE OF STUDY

From the observations and conclusions made by Miller, as reviewed above, it is significant to note that advanced alveolar bone loss may have its initiation between 5 and 13 years of age. The actual detection of this condition, however, has not been reported during this early period when the permanent teeth are erupting. In a study of periodontal disease in children, Zappler[22] states that retrogressive changes such as periodontosis and alveolar atrophy are rarely found in healthy children. She also points out, however, that when puberty occurs there is a decided increase in periodontosis and alveolar atrophy in healthy individuals. In such adolescent patients with periodontosis it can be assumed that the pathologic process may date back to an earlier age. The cause of atrophic, degenerative, and retrogressive changes in the periodontal structures of a young person are obscure at this time.

It is clear that Zappler's observations do not preclude the possibility of this disturbance existing in apparently healthy children. Consequently, there is need for a statistical study to determine whether there are early signs of retrogressive alveolar bone changes in such individuals. These same children show alveolar changes upon reaching puberty or later in life while still apparently in a state of good health. The findings, whether they be positive or negative, may suggest a path of study to determine the nature of such manifestations. By correlating all factors in the background of the cases studied, some objective may be brought into sight.

METHODS AND MATERIALS

The roentgenograms of patients in the Department of Pedodontics at New York University College of Dentistry were studied. Children no longer are treated in this clinic when all the deciduous teeth have been exfoliated. Those roentgenograms were selected for study which showed the presence of erupted permanent teeth, regardless of number. The following information was recorded from each patient's chart so that the material could later be correlated with the roentgenographic findings: 1) name 2) date of birth 3) sex 4) temperament 5) soft tissue condition 6) habits 7) occlusion 8) diet 9) medical history.

So as to properly interpret the roentgenographic appearance of the periodontium (bone, periodontal membrane, cementum), it is necessary to have a knowledge of the differences in the anatomy of these structures as compared with those of adults. Such a description was obtained from Zappler's study.[22] In newly erupted teeth of children there is only a thin layer of cementum. It's calcification is less dense. The thickness of the periodontal membrane is greater in young individuals. The fiber bundles of the periodontal membrane are less dense during the eruption process and until the teeth are able to function. There are more loose and irregularly arranged fibers and fewer collagenous fibers. The blood and lymph supply are more abundant in the periodontal membrane of the young person. The mobility of the teeth during eruption is not pathologic, but is due to the existent state of structure of the periodontal membrane and to the incomplete formation of the roots. In this stage of development, the lamina dura of the alveolar bone is thinner. The trabeculae in the supporting bone are fewer and the calcification is less dense. The blood and lymph supply is more abundant. The alveolar crests in the newly erupted permanent teeth are more pointed than in the adult. In the deciduous teeth, the alveolar crests are flatter. The bone is more plastic. These factors should be taken into consideration when making this study, for they are all encountered in patients with mixed dentitions. It is obvious that we cannot utilize the incipient signs of periodontal disease in adults in evaluating the roentgenographic findings. Instead, the existing height of the alveolar bone would have to be used as an indicator of disturbances in the bone. In order to adequately measure the amount of alveolar bone resorption, it was found that the method advocated by Miller and Seidler[23] in their study of the relative alveoloclastic experience of the various teeth would be suitable as a basic guide. Each erupted permanent tooth would be given a number ranging from 0 to 5. When there was no bone resorption, a unit of 0 would be given to the tooth. A unit of 3 was given a tooth when the bone destruction had left one half the root devoid of bone. Units 1, 2, and 4 were estimated as fractions of 3 and 5. Even with this method it is easily seen that difficulties would arise in accurately differentiating various levels of bone when exfoliating deciduous teeth or erupting permanent teeth are adjacent to already fully erupted permanent teeth. At best, in some cases, only gross deficiencies of bone would be distinguished. In spite of these difficulties, it was decided to carry out the intended studies so that a trend might be noticed or other procedures suggested. In view of these facts, the survey should be considerd as a "pilot" study from which we may profit for future investigations.

FINDINGS

The roentgenograms and records of 89 patients were studied. Of this number, 52 were males and 37 females. Most patients were in the age group of 7-9 years, the exact numbers being as follows: 7 years-19 patients; 8 years-17 patients; 9 years-22 patients. Except for the 12 and 13 year old groups, males predominated in each age group. A tabulation of the many factors in the medical and dental backgrounds of these patients (Table I) revealed certain interesting relationships. 34 patients were considered nervous and 55 were described as being of normal temperament. 48% of the total number of males were found to be of the nervous type, while only 24% of the total number of females were found to be of the same tempera-

TABLE I

	6		7		8		9		10		11		12		13		TOTALS
Age																	
No. of Cases	10		19		17		22		11		6		3		1		89
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	
	6	4	14	5	9	8	12	10	6	5	4	2	1	2	0	1	MALE 52
																	FEMALE 37
TEMPERAMENT																	
Nervous	3	1	6	1	4	1	6	2	4	3	2	0	0	1	0	0	34
Normal	3	3	8	4	5	7	6	8	2	2	2	2	1	1	0	1	55
SOFT TISSUES																	
Normal	6	4	14	5	9	8	11	10	5	5	4	2	1	2	0	1	87
Gingivitis	0	0	0	0	0	0	1	0	1	0	0	0	0	0	0	0	2
HABITS																	
None	3	2	7	1	5	6	7	4	5	1	2	2	0	1	0	1	47
Bruxism	1	0	3	0	0	0	1	1	0	0	0	0	0	0	0	0	6
Finger Sucking	0	2	0	1	1	0	2	4	0	1	0	0	0	0	0	0	11
Nail Biting	2	1	5	3	2	2	5	4	1	3	2	0	1	1	0	0	32
Lip Biting	1	1	1	2	2	0	2	0	1	0	1	0	0	1	0	0	12
Tongue Habit	0	0	1	0	0	0	0	1	0	0	0	0	0	0	0	0	2
OCCLUSION																	
Normal	5	1	11	3	7	6	8	6	4	3	2	2	0	2	0	1	61
Abnormal	1	3	3	2	2	2	4	4	2	2	2	0	1	0	0	0	28
DIET																	
Adequate	6	3	10	4	7	5	11	9	3	5	4	2	1	2	0	1	73
Soft	0	0	1	0	1	1	0	0	0	0	0	0	0	0	0	0	3
Carbohydrate	0	1	2	1	1	2	1	0	1	0	0	0	0	0	0	0	9
Avitaminosis	0	0	1	0	0	0	0	1	2	0	0	0	0	0	0	0	4
MEDICAL HISTORY																	
Measels	5	3	10	5	4	6	5	3	4	2	4	0	1	2	0	1	55
Mumps	0	2	4	2	1	2	4	2	2	3	2	0	0	1	0	1	26
Chicken Pox	3	2	4	2	3	4	9	4	5	2	2	0	0	2	0	1	43
Whooping Cough	0	0	1	0	5	1	3	1	2	1	0	1	0	0	0	0	15
Rheumatic Fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Pneumonia	0	1	0	1	0	0	0	0	0	1	0	0	0	0	0	0	3
Meningitis	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1
Scarlet Fever	0	0	0	1	0	0	0	1	0	0	0	0	0	1	0	0	3
Allergy	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	2
Poliomyelitis	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	2
Asthma	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	1
Anemia	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
Diphtheria	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1
ALVEOLAR BONE CONDITION																	
Amount																	
0	6	4	14	5	9	8	12	7	6	4	3	2	0	2	0	1	83
1	0	0	0	0	0	0	0	1	0	1	1	0	1	0	0	0	4
2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
5	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other	0	0	0	0	0	0	0	2	0	0	0	0	0	0	0	0	2

M = Male F = Female

ment. Only 2 of the total number of cases studied were listed as having gingivitis. The others were considered normal. More than half the patients were found to be free of habits. In order of frequency, the most common habits were nail-biting, lip biting, finger sucking, bruxism, and tongue habits. On the basis of the total number of males and females studied, greater percentages of males were found to have a lip biting habit and bruxism. On the other hand, greater percentages of females demonstrated finger sucking and nail biting habits. The existence of these habits according to age were in proportion to the original distribution. The occlusion was abnormal in 28 cases of the 89 investigated. This number constituted 28% of the total number of males and 35% of the total number of females. It was revealed that 73 cases had an adequate diet while 9 showed evidence of a high carbohydrate diet. In the medical backgrounds of the cases studied, measles, chicken pox, mumps, and whooping cough occurred most often in both sexes. An evaluation of the roentgenograms of these cases showed that 83 did not manifest any apparent signs of alveolar bone disturbances. Each of these cases was assigned a unit of 0, according to the system of classification previously described. Six cases (6.7% of the total) presented roentgenographic signs suggestive of alveolar bone changes. Four of these were assigned a unit of 1 since the level of the alveolar bone was in question. Two of the cases presented other facts which warranted consideration as possible signs of abnormality of the alveolar bone.

Each of the six cases considered abnormal was analyzed for a more precise interpretation of the findings. The object was to determine whether the roentgenographic signs were actually indicative of retrogressive alveolar bone changes. In this manner certain standards for evaluation may be set up. Pitfalls which may incorrectly influence our decisions may thus be avoided.

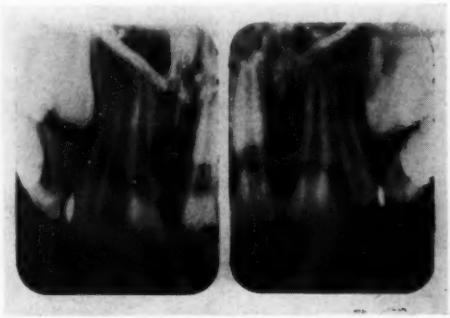

Fig. 1—Case No. 1, female, age 9. Note the radioluscency of the alveolar bone between the central incisors.

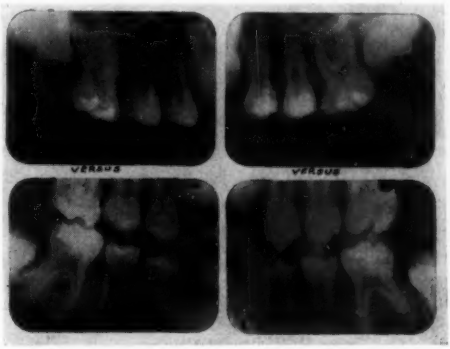

Fig. 2—Case No. 2, female, age 9. Compare the height of the buccal or lingual plate of bone of the maxillary left and right premolars in the periapical films with that in the bitewing films below.

Case #1 (Fig. 1), female, age 9, demonstrated a diffuse radioluscency of the alveolar bone between the maxillary central incisors. The alveolar crest was not well defined. On close examination, however, a continuous lamina dura could be seen. It is possible that the trabeculae were obliterated by overexposure. The habits existing in this case were finger sucking, finger nail biting, and a tongue habit. Previous diseases included measles, mumps, and chicken pox. A Class II malocclusion existed. The temperament and soft tissues were considered normal and the diet adequate.

Case #2 (Fig. 2), female, age 9, showed a deficiency in the height of either the buccal or lingual plate of bone of the maxillary pre-

molars. In the bitewing film of this same area, though, the alveolar height appeared normal. Differences in angulation must be considered in the interpretation of these two roentgenograms. In addition, the premolars in question have not erupted completely. Habits did not exist, the medical history was negative, and the diet was adequate. Other factors were normal.

Case #3 (Fig. 3), female, age 9, revealed a radioluscency of the alveolar bone in the upper anterior region. A hazy alveolar crest was also present. Such a picture was in complete contrast to roentgenograms of the same area taken

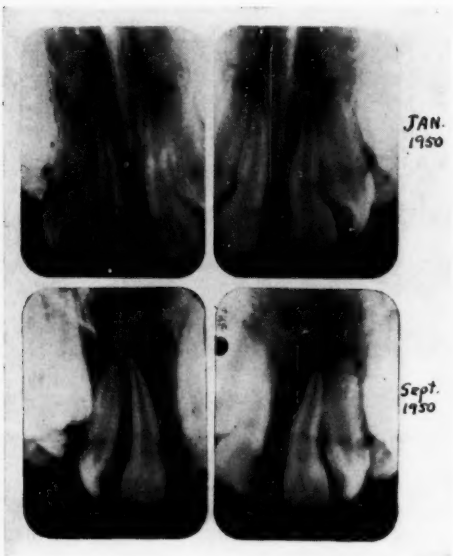

Fig. 3—Case No. 3, female, age 9. Note the radioluscency of the alveolar bone and the hazy alveolar crests in the more recent periapical films as opposed to the condition existing 9 months before.

9 months before. Here again, the differences may be accounted for on the basis of angulation and exposure time. Habits of this patient included finger nail biting and finger sucking. The individual was considered nervous. Avitaminosis existed and the occlusion was abnormal. Chicken pox and measles were diseases previously encountered. The soft tissues of the mouth appeared normal.

Case #4 (Fig. 4), female, age 10, had what appeared to be a deficiency in the height of the labial or lingual alveolar plate of bone in the maxillary lateral incisor regions. The canines could be seen erupting adjacent to these teeth. Finger sucking existed and there was an open bite anteriorly. Previous diseases included measles and mumps. The diet was adequate. Other factors were normal.

Case #5 (Fig. 5), male, age 11, showed a radioluscency and resorption of the alveolar crest between the maxillary central incisors. The two central incisors had been fractured near the incisal edges. A roentgenogram taken one year before revealed only one central incisor to be fractured and the absence of any radioluscency. It would seem as though the roentgenographic changes could be directly

Fig. 4—Case No. 4, female, age 10. Note the height of the labial or lingual alveolar plate of bone in the lateral incisor regions and the position of the erupting canines.

related to the effects of severe trauma. Fingernail biting and lip biting were present. There was abnormal occlusion posteriorly. The individual was of the nervous type. The diet was adequate and the soft tissues normal. Previous diseases included chicken pox, measles, and mumps.

Case #6 (Fig. 6), male, age 12, roentgenographically showed the labial or lingual plate of bone below normal height in the maxillary right central and lateral regions. In the same area on the left side the level of bone appeared normal. The incomplete eruption of the maxil-

lary right canine must be considered as a possible underlying cause. Finger nail biting was the only habit present. A cross-bite relationship existed posteriorly. The diet was adequate. The only childhood disease was measles. Other factors were normal.

Of the six cases described, four were females and two were males. In five of these six cases of suspected or questionable alveolar bone changes, habits such as finger sucking, finger nail biting, and tongue habits existed. The diet was adequate in five of the six cases. The most common childhood diseases of chicken pox, mumps, and measles were encountered in all but one of these cases. The temperament of two of the six cases was of the nervous type. The soft tissues were normal in all of the cases. Abnormal occlusions were present in all but one case.

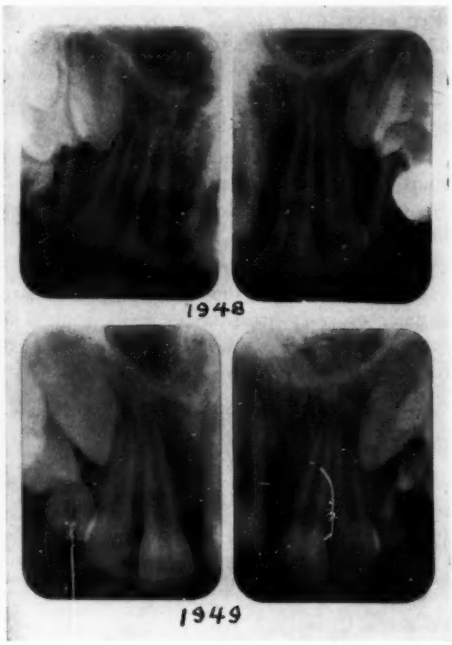

Fig. 5—Case No. 5, male, age 11. Compare the increased radioluscency and resorption of the alveolar crest between the central incisors in the more recent periapical films with that in the earlier films. Note also in the earlier film that only one central incisor is fractured.

DISCUSSION

In evaluating the roentgenograms of children for retrogressive alveolar bone changes, there appear to be several factors which must be borne in mind. Due to the transitional stage of the dentition, roentgenographic signs may be present which simulate adult periodontal disease. The erupting permanent teeth bring along a new alveolar process which undergoes progressive stages in development. This is most likely the reason for the buccal or lingual plates appearing below normal level on incompletely erupted teeth or adjacent to teeth which are erupting. The cortical plate of bone becomes evident at the alveolar crest in the later stages of development. It is absent when the teeth first erupt. This may thus give the appearance of alveolar crest resorption and radioluscency. The

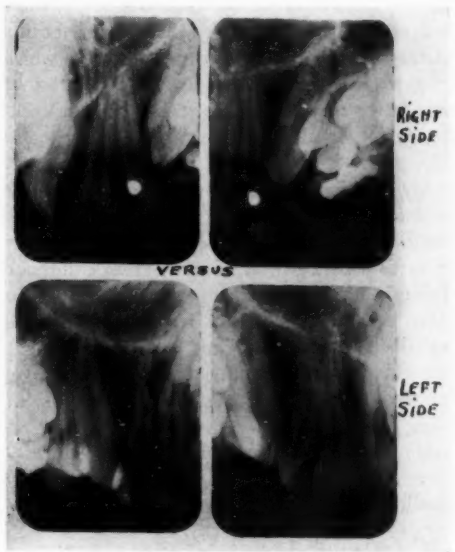

Fig. 6—Case No. 6, male, age 12. Note that the height of the labial or lingual plate of bone in the right lateral and central incisor regions is below that on the left side. Also note that the right canine has not erupted as far as the left canine.

variation in the angulation of the X-ray tube may change the appearance of structures from one roentgenogram to another. Overexposure of films is another error which may be misleading in the interpretation of roentgenograms. The realization of such possibilities will prevent one from prematurely arriving at a conclusion. Thoughtless decisions which are made after only a superficial study can be misleading and disastrous. Inferences should be based on indisputable facts.

Certain conclusions may be drawn from this study which may be of benefit in the future. On the basis of the findings in this study, there still remains the possibility that a predisposing factor may be present during childhood which may weaken the bone structure. The effect of this factor may remain dormant until an exciting factor, local or systemic, occurring later in life, causes it to be manifested. This most certainly deserves further investigation. It is quite evident that a more controlled study is indicated. This would involve a continuous study of cases over a period of years from childhood through adult life. Roentgenograms would be taken at regular intervals. In effect this would provide a serial study. Only in this manner can one expect to thoroughly analyze any changes in alveolar bone. A much larger number of cases must be included in the study if any reliable conclusions can be drawn. Uniform techniques in roentgenology and history taking should be a primary goal. Finally, it would be beneficial if a comprehensive set of criteria could be decided upon for a more accurate evaluation of roentgenograms in children.

Grateful acknowledgment is made to Dr. Samuel Charles Miller, Chairman, Department of Periodontia, New York University College of Dentistry; Dr. Harold K. Addelston, Chairman, Department of Pedodontics, New York University College of Dentistry; and to Dr. S. Sigmund Stahl, Research Associate, Department of Periodontia, New York University College of Dentistry, for their advice and guidance in the preparation of this study.

Thanks are also extended to Dr. Addelston for making available the records in the Department of Pedodontics, New York University College of Dentistry.

SUMMARY

1. A review of the literature on the etiology of precocious advanced alveolar atrophy and periodontosis was presented.

2. In order to shed some light on the etiology of this type of periodontal disease, a pilot study of the roentgenograms of 89 children between the ages of 6 and 12 was made. The objective was to determine early roentgenographic signs of retrogressive alveolar bone changes and to correlate all other factors in the background of these individuals.

3. Six cases presented roentgenographic signs suggestive of alveolar changes. An analysis of these cases made evident certain limitations in interpretation. As a result, there was inconclusive evidence of such changes.

4. Future studies should include a greater number of cases made under more controlled conditions. More desirable criteria are needed for correct evaluation of roentgenograms.

132 East 72nd Street, New York, N. Y.

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NEW YEAR

Medical Views on Oral Problems

HERPES SIMPLEX INFECTIONS*

Primary herpetic gingivostomatitis has come to be recognized as one of the common infectious diseases of childhood. The virus of herpes simplex was first implicated as the etiological agent in 1938 by Dodd, Johnston and Buddingh. Buddingh and associates review studies pursued during the course of several years in the pediatric clinics and wards of Vanderbilt University Hospital. The virus of herpes simplex was isolated and identified and the specific serologic response to primary infection was demonstrated in 52 children ranging from 1 to 3 years of age. These cases provided the material required for the development of techniques and methods. In a detailed study of 12 patients the virus was recovered from the mouth and stools during the acute stage, convalescence, and for varying periods thereafter. The average time during which virus could be recovered from these sources was 23 days after the onset of the disease. Virus could not be recovered from the blood or spinal fluid, but patients were not available for study before the third day of the disease. Neutralizing antibodies to herpes simplex were first demonstrated in the serum from the fourth to seventh day of the disease. The antibodies attained a maximum level during the second or third week. The maximum antibody concentration was not maintained and dropped to relatively low levels over varying periods. Herpes simplex virus was recovered from the mouths of persons presenting no clinical evidence of herpetic infection; it was recovered from 7% of 571 normal individuals of various ages; from one infant out of 97 under 6 months of age, from 20% of 72 children of 7 months to 2 years, from 9% of 199 children 3 to 14 years of age, and from 2.5% of 185 adults. Neutralizing antibodies were demonstrated in the serum of 43% of white and 71.4% of Negro children in the 7 month to 2 year age group. In adults 90% of white and 96% of Negro individuals had antibodies to herpes in the serum. Natural active immunity appears to be established at an earlier age in the segments of the population in which the highest carries rate was demonstrated. Neutralizing antibodies were demonstrated in the serum of 76% of white infants and in 90% of Negro infants 6 months of age and under. The placental transmission of neutralizing antibodies was demonstrated. Approximately the same level of antibody was found in the maternal and placental serums.

* Medical Literature Abstracts, J.A.M.A., Oct. 3, 1953.

HYDROCORTISONE (COMPOUND F) BY LOCAL INJECTION IN ARTHRITIS*

Ramsey and Key present their observations on 119 patients with arthritis who received a total of 218 local injections of hydrocortisone. These patients had not received adrenal steroids by any other route, and they were observed for a sufficient period of time to permit evaluation of the results. The injections were given after 1% procaine had been injected into the skin and along the path of the needle but not into the joint, if this could be avoided. The Hydrocortisone was then injected into the joints with another needle and syringe. The dose of hydrocortisone was from 25 to 50 mg. for the major joints and 5 to 20 mg. for the smaller joints. A total of 100 injections was given into 57 joints of 51 patients with hypertrophic arthritis. The response was considered satisfactory if there was at least a 50% relief in symptoms for a minimum of a week. With this criterion, 48 of 57 joints showed a satisfactory response. Many of these patients experienced nearly complete relief of symptoms for from 3 to 10 weeks or longer. In 14 patients with active rheumatoid arthritis 20 of 22 involved joints showed some response to intra-articular injections of hydrocortisone, but in general the response in these patients was of shorter duration than was the case in patients with hypertrophic arthritis. Thirty-three patients were given a total of 52 injections into painful bursas, with curative effect in 17 (mostly acute calcific bursitis of shoulder), partial relief in 9, and no benefit in 7. Tennis elbow, painful heels, acutely inflamed bunions and hemarthrosis of the knee following meniscectomy were some of the other conditions in which hydrocortisone was administered by local injection. No serious complications were observed in these patients, but the possibility of producing aseptic arthritis by contamination should be kept in mind, and a careful injection technique should be used. The authors resorted to intra-articular injection only in selected cases with relatively severe symptoms, because they feel that a needle should not be introduced into a joint without good reason. While the treatment is not a panacea, it is a valuable therapeutic agent not only for acutely painful arthritic joints but also for its local anti-inflammatory action in several other painful conditions in which the etiology is obscure.

* Medical Literature Abstracts, J.A.M.A., Oct. 24, 1953.

CHLOROPHYLL AS A DEODORANT*

To the Editor:—I have recently seen an article in a professional journal on chlorophyllin products that seems to me to contain misleading and unreliable information. Some of this information pertains to advertising and can be used for the promotion of marketed preparations.

First, of all, the material sold as chlorophyll is not chlorophyll at all and does not even merit the name chlorophyllin. Chlorophyll is a complex molecule containing the three important portions: (1) the group of four pyrrole rings, (2) the side-chains esterified with phytol and methyl alcohol, (3) the magnesium atom in the center. On treatment with alkali, the phytol and methyl alcohol are removed, and the remaining salt of the free acid is called a chlorophyllin. On subsequent treatment with acid, the magnesium is removed and the residue is called a pheophorbide. If the magnesium is removed first, the residue is a pheophytin. The pheophytins are olive-brown but become green when a molecule of copper or nickel is added. To the best of my knowledge, the commercial preparations are copper or nickel pheophytins or pheophorbides. In the article in question, the term "complex processing" merely refers to this successive treatment with alkali, acid, and a metal salt. This is far from complex and only gives a false impression of difficulties in manufacture.

I understand that most of the commercial products involve the use of one group of patents. I am not familiar with the wording of these patents, but if, as I believe, they refer only to the preparation of water-soluble materials from chlorophyll, I must point out that these reactions were studied early in the 20th century by Schlunk and Marchlewski and by Willstätter. A particularly full account is given in Willstätter. "Untersuchungen über Chlorophyll," which was translated into English by Schertz and Merz and published in 1928. To my knowledge, the American Chlorophyll Company has been producing water-soluble pheophytin preparations for more than 10 years.

It is important to insist on the correct use of names because the word chlorophyll, or even chlorophyllin, gives a false impression to the public. Most persons associate the virtues of these prepared chlorophyll products with the known efficiency of chlorophyll in carrying out photosynthesis. This material is not chlorophyll, and, even if it were chlorophyll, it would not be in a state of biological activity. Chlorophyll, as it occurs in the leaf, is combined with proteins in the chloroplast, and it is only in that form that it is photosynthetically active.

By far the most misleading part of the article is the reference to tests that are supposed to have proved chlorophyll effective in deodorizing and in large numbers of other activities. The status of these tests is extremely doubtful. In the first place, it is hardly necessary to point out that the quantitative determination of the intensity of smell is a very difficult undertaking. As a rule, odors are compared by professional taste and odor experts who use a series of reference solutions that are made up in standard concentrations and that operate in as nearly as possible a reproducible way. It is quite out of the question to apply these procedures, rough and empirical as they are, to wounds and persons' breath or body odors. It follows that the so-called tests are very likely to be valueless. The publications in which I have seen descriptions of experiments of this sort make little or no mention of any precautions to obtain reliable data as to the intensities of the odors that they claim are removed. I have seen no reliable evidence to indicate the effectiveness of chlorophyll in any of the activities claimed for it.

It is true that I have by no means studied all of the literature dealing with the subject and that one can always draw a false impression from what is written. Let me say that I will be happy to withdraw any or all of the above statements if anyone can show me convincing evidence that chlorophyll acts as a deodorant or a cure for gingivitis or that it has any greater effect in promoting the growth of granulation tissue in wounds than do a vast variety of other nonspecific materials. It is my impression that the public is being subjected to a racket of tremendous proportions and that the pharmaceutical industry is collaborating in this.

KENNETH V. THIMANN, Ph.D.
Professor of Biology
Harvard University
16 Divinity Ave.
Cambridge 38, Mass.

* Correspondence, J.A.M.A., June 27, 1953, p. 869.

DEATH FOLLOWING THE USE OF EFOCAINE*

By Alden L. Angerer, M.D., Hung H. Su, M.D., and Jerome R. Head, M.D., Chicago.

Intercostal injection of Efocaine (1.5cc) was followed by death of the patient in 44 hours. Efocaine is a local anesthetic with a prolonged action that has been used to control prolonged pain, neuralgias, and itching. It consists of a solution of 1% procaine, 0.25% procaine hydrochloride, and 5% butyl-p-aminobenzoate in a solvent composed of 2% polyethylene glycol-300, 78% propylene glycol, and water. Previous reports of transverse myelitis, toxic neuritis and other complications to its use have also accumulated. The authors conclude that it should not be used for local anesthesia.

* J.A.M.A., Oct. 10, 1953, Vol. 153, No. 6, p. 550. Abstracted by Arthur N. Yohai.

USE AND ABUSE OF CORTISONE AND CORTICOTROPIN (ACTH) IN DERMATOLOGY*

By Paul A. O'Leary, M.D. and John G. Erickson, M.D., Rochester, Minn.

The authors review their considerable experience with ACTH and cortisone in dermatology and find the results discouraging and often dangerous. Early promising cures have often proved to be short remissions with ensuing serious relapses, or side reactions of significant proportions. They limit the use of these drugs to very few dermatologic conditions under very careful supervision.

(Editor's note—This would seem to be further confirmation of the now recognized attitude that local or systemic administration of these drugs in dental practice is unwarranted.)

* J.A.M.A., Aug. 29, 1953, Vol. 152, No. 18, p. 1695. Abstracted by Arthur N. Yohai.

FILTERABLE AGENT CAUSING MOUSE SALIVARY GLAND CARCINOMA*

Gross, working with mice of the AK inbred line who have a tendency to develop leukemia "spontaneously", has isolated a filtrable agent which is the apparent causative factor when transmitted directly to the embryo of other strains as well as (C3H). On further attempting to fractionate the extract containing this factor through finer filters, the results were surprising. 9 of the 84 inoculated animals developed typical leukemia. However 15 others developed bilateral salivary gland carcinomas instead, which had never been discovered before in either strain. This would seem to indicate that more than one oncogenic agent may be passed at one time, and that one can repress the action of the other.

* J.A.M.A., Sept. 12, 1953, Vol. 153, No. 2, p. 150. Abstracted by Arthur N. Yohai.

FATAL FUNGUS INFECTION COMPLICATING ANTIBIOTIC THERAPY*

By Curtland Brown, Jr., M.D., Simon Propp, M.D., C. Maynard Guest, M.D., Richard T. Beebe, M.D., Albany, N. Y. and Lawrence Early, M.D., Hudson, N. Y.

Five cases terminating fatally are reported, two of them dentists. All had received heavy antibiotic (combination) therapy for severe systemic illness. In every case Candida albicans was implicated. In one or more cases other fungi, yeasts, molds, or Mycobacterium tuberculosis were involved as well. The authors conclude that patients who are receiving broad spectrum antibiotics should be carefully observed for evidence of fungus invasion.

(Editors note — This should constitute warning against the indiscriminate dental use of antibiotics without sufficient cause.)

* J.A.M.A., May 16, 1953, Vol. 152, No. 3, p. 206. Abstracted by Arthur N. Yohai.

Book Reviews

Orthodontics, Diagnosis - Prognosis - Treatment. By Bercu Fischer, D.D.S., W. B. Saunders Company, Philadelphia and London, 1952. 334 pages, 1180 illustrations on 212 figures. $12.00.

Dr. Fischer has written a carefully conceived and copiously illustrated book on clinical orthodontics. The basic theme of the book may be summed up by a quotation from Chapter II, "The Individuality Hypothesis". "Natural variation is recognized as a universal basic attribute of the human body; thus variation has its beginning in heredity; the compensatory adjustment mechanism fits the variable parts together into a unique morphologic unit. Starting with this mosaic individuality, functional adaptation through the dynamic and random forces gives the dentofacial complex its specificity by further establishing the individuality of its functional pattern."

The clinical material presented illustrates the basic theme and substantiates the Individuality Hypothesis. The concepts of orthodontic Normal standards are reviewed to show the incongruity between statistical normals of all types and their application to the individual dentofacial complex. Hazards of orthodontic treatment are described and their clinical remedy illustrated. Root resorption and instability of the treated denture being the major hazards.

The last four chapters are devoted to the technical procedures required to attain the objectives of treatment. The use of occipital force replaces intra oral reciprocal force. Sectional treatment replaces en masse movement of teeth, early treatment in the mixed dentition in a large percentage of cases reduces the number of cases needing extraction in treatment. The concomitant case reports in the book illustrate this approach to treatment with large clear photographs. While this work is of primary importance to the practitioners of orthodontia, the chapters on "The Individuality Hypothesis", "The Meaning of the Normal" and "Methods of Recording" will be of interest to the Dental profession as general practitioners or as practitioners of the various specialties.

A Text-Book For Dental Assistants. By Irwin Robert Levy, D.D.S., Third Edition, Lea & Febiger, Philadelphia. 272 pages, 232 illustrations. $4.00.

This third edition of a previous work brings the text generally more up to date, and has appended a dental dictionary of 1500 commonly used terms. The first 82 pages are concerned with chair side assisting and dental office management. These are adequately covered, although one could hope for a little more consideration of dental techniques, materials and their properties. The section on the psychology of patient office relation and practice administration is particularly apropos. Other chapters are concerned with dental anatomy, sterilization, dental radiology, oral surgery, periodontia, oral hygiene, pedodontia, and orthodontia. A brief consideration is given to impression taking and making models, and a separate chapter is devoted to gold inlays and acrylic crowns.

This text has a definite place in any office that employs assistance, and would serve as a primer in the indoctrination of a dental assistant.

ARTHUR N. YOHAI

Inlays and Abutments. By Jacob R. Schwartz, D.D.S., Dental Items of Interest Publishing Co., Inc., Brooklyn, New York, 478 pages, 514 illustrations. $8.00.

As is indicated by the title, this book deals exclusively with the preparation of teeth for receiving both inlays and other type restorations where these teeth serve as abutments.

In preparing the reader for the technical aspect of the book, the author has given practical consideration to defining his terms, setting up his classifications and to discussing various types of instruments. This is a beneficial plan since the practitioner can then follow the techniques described by Dr. Schwartz.

Preparations for each tooth are discussed in detail and in many instances methods of other operators are compared to those given by the author. Details include step by step procedure in the actual completion of the operation. These descriptions are enhanced by numerous detailed illustrations. Indications for the use of the various types of preparations and restorations are given throughout the text.

The last portion of the book is devoted to technical details of impression taking, die making, electroplating, use of hydrocolloids, casting and types of gold to be used.

The book is well planned, well written and the

publishers have used bold type on coated paper which makes the reading easy. The illustrations are good and well reproduced. This book is recommended reading for both students and practitioners. It is a worthwhile addition to the dental literature.

LOUIS R. BURMAN

Noyes' Oral Histology and Embryology. By Isaac Schour, B.S., D.D.S., M.S., Ph.D., Sc.D. 7th edition, Lea and Febiger, Philadelphia, 1953. 448 pages, 530 illustrations. $8.00.

As the knowledge of the etiology of periodontal disease increases, it is evident that careful attention to etiology in the early stages will prevent much destruction of the periodontium. One of the earliest signs of periodontal disease is the thickened periodontal membrane as seen on the roentgenogram. Dr. Schour writes that "both the structure and the width of the periodontal membrane are determined by its functional condition. It is thickest at the alveolar crest, thinnest below the middle of the root and wider again towards the apex. When subjected to normal masticatory stress the width averages 0.20 to 0.25 mm. and its structure is normal as described above. If the tooth is subjected to increased functional stress due to the loss of adjacent teeth, the width increases to 0.28 to 0.35 mm. The structure becomes modified also, the fiber bundles becoming stronger and the loose connective tissue being greatly reduced. This picture is typical of functional hypertrophy. If, on the other hand, the tooth lacks in functional stress due to the loss of its antagonist, the average width is reduced to 0.10 to 0.15 mm. The fiber bundles atrophy and are replaced by loose, irregular connective tissue without definitive functional orientation." This explains why a thickened periodontal membrane is an incipient sign and the alert practitioner can do much for the patient at this stage.

In each chapter the author has included a heading titled Histophysiological remarks. In these sections oral histology is made vital or physiologic. Not only does he explain why a particular structure is so constructed, but also how it functions both in health and disease. The text is more than just an oral histology book for the dental student; it is a practical guide for the practicing dentist.

Dr. Schour has spared no efforts in his illustrations. He has supplemented the photomicrographs and drawings of previous editions with new ones, many in color. This certainly adds a great deal to prove that what he writes is not just theory but fact. It seems that every illustration is documentary proof of the text material. It would be possible to learn a great deal just by studying the illustrations and the captions.

For those dentists who care to, and those students who use this text as the text of oral histology there is a second section in the book which explains the directions for laboratory work.

NATHAN WACHTEL

Dental Anatomy. By Moses S. Diamond, D.D.S. 3rd edition, Macmillan, New York, 1952. 471 pages, 300 illustrations. $15.00.

This is the third edition of the late Dr. Moses Diamond's book. Being one of the standard texts on dental anatomy it might be thought that a new edition would have only minor revisions. This manuscript, completed a short time before his death is full of many important changes and additions.

The second half of the book is devoted to an atlas of the head and neck. Here gross anatomy of the head and neck is presented in a manner that makes it quite easy to understand. Series A in this group of full page illustrations depicts progressively deeper dissections of the same cadaver. This takes the surgeon, lead by the anatomist, to the operating table where he can review the dissection layer upon layer just as he might do with a patient.

Series B illustrations start with a plate showing radial sections through the left side of the face. The following 18 illustrations are the actual radial sections. This makes it quite easy to follow.

In the section on tooth morphology, Dr. Diamond carefully describes the manner of reproducing teeth in wax. As dental educators know, this is the best way of learning dental anatomy. Each tooth is described and profusely illustrated.

Of importance and value in any text book is the practical value of the knowledge presented. This book is certainly full of the applications of dental and gross anatomy. There are chapters on temporomandibular articulation, the vascular system, the nervous system, and the oral cavity and its soft tissues. Each one relating the anatomy to dental practice.

In reviewing this encyclopedia, the fact that the author describes the development of each structure mentioned from its inception to maturity, must not be overlooked. For example, in the temporomandibular joint there is a description of the infant's joint and how it is related to its manner of sucking. The further development is discussed with illustrations, depicting all stages to the full development of the temporomandibular joint in the beginning of the third decade.

There are some truly remarkable illustrations of human anatomy of the head and neck in early infancy and childhood.

NATHAN WACHTEL

Textbook of Exodontia. By Leo Winter, D.D.S., M.D., LL.D., and revised by William Harrigan, A.B., D.D.S., M.D., and Leo Winter, Jr. D.D.S.

The C. V. Mosby Co., St. Louis, 1953, Sixth Edition, 350 pages, 385 illustrations and one color plate. $8.00.

This sixth edition is a revision of the previous editions of a "Textbook of Exodontia" by the late Leo Winter. It is intended primarily as a text for dental students and general dental practitioners. The chapters dealing with chemistry of anesthetic agents and general anesthesia have been deleted. New chapters have been added which include: Diagnosis and History Taking; Chemotherapy and Antibiotics; Hemorrhage; and Alveoloplasty. The remaining chapters are basically the technics as presented in the other editions.

The book is well illustrated with pictures, drawings and cuts of radiographs. The general practitioner would benefit greatly from this book because it not only encompases routine surgical procedures, but deals with difficult cases often sent to a specialist.

Oral Anatomy. By Harry Sicher, M.D., D.Sc. The C. V. Mosby Company, St. Louis, Second Edition, 1952. 529 pages, 310 illustrations including 24 in color. $13.50.

This book is an excellent text for physicians and dentists who wish to review their knowledge of head and neck anatomy. In this second edition, Dr. Sicher has incorporated new facts and concepts that were not accepted when the first edition was published. Many inconsistencies of nomenclature have been eliminated. In many chapters the boundaries of pure anatomy have been transgressed where for instance surgical technique is touched upon to show that it can be developed as a logical consequence of anatomic study.

The text is divided into two parts; part one on descriptive anatomy, and part two with regional and applied anatomy. There are many chapters that are of special interest to the dental practitioner. Chapter 3 deals with the Temporomandibular Articulation, its anatomy and movements, Chapter 4 reviews the Viscera of the Head and Neck, while Chapter 7 gives an excellent discussion of the Nerves of the Head and Neck.

In the field of regional and applied anatomy, Chapter 10 discusses the Anatomy of Local Anesthesia, while Chapter 12 covers the Propagation of Dental Infections. Temporomandibular Articulation, overclosure and distal displacement, dislocation and surgical exposure are explained in Chapter 14, while Chapter 15 covers the Edentulous Mouth.

The book is well illustrated with black and white drawings and 24 plates in color. The type is clear and large and makes for easy reading| It is an excellent reference volume for the dental practitioner and student.

IRVING YUDKOFF

Problems in Dental Local Anesthesia. By Mendel Nevin, D.D.S., and revised and edited by Hillard R. Nevin, D.D.S. Dental Items of Interest Publishing Co., Inc., Brooklyn, New York, 760 pages, 274 illustrations. $10.00.

The authors have produced a volume on dental anesthesia that covers just about every clinical phase of the subject. With the help of over 90 contributors from this country and abroad, the usual questions that dental practitioners ask are anticipated and answered.

The text covers local anesthetics, vasoconstrictors, armamentarium, sterilization, and fully discusses the many types of injections. In addition, there are chapters dealing with pre and postoperative medication, antibiotics, and sulfonamides, broken needles, failures in local anesthesia, and complications of dental anesthesia.

PROBLEMS IN DENTAL LOCAL ANESTHESIA is a volume of almost eight hundred pages with hundreds of illustrations covering phases of dental local anesthesia. It is recommended as a reference book and should be in the library of the general practitioner.

IRVING YUDKOFF

Periodontology: Diagnosis and Treatment. By Frank E. Beube, D.D.S., New York. The Macmillan Company, 1953. 752 pages with 492 photographic and line illustrations. $15.00.

The text consists of twenty-six chapters covering all phases of periodontia, including etiology, pathology, diagnosis and treatment. Also included are many fine illustrations as well as an extensive bibliographical and suggested reading list.

The book cites the confusion associated with periodontitis versus periodontosis, and includes a table of differential diagnosis for the convenience of the reader.

The author mentions the difficulty in obtaining repair in cases of advanced periodontal breakdown around single rooted teeth and describes the work done and the results obtained by the use of boiled cow-bone powder.

Dr. Beube states the following concerning diet and nutrition: "At the present time, well-controlled clinical research on patients has failed to provide proof that any given substandard diet has a specific deleterious effect on the periodontal tissue. Furthermore, the unbalanced diets eaten by many or perhaps by most patients have not been shown to be related to the various gingival and periodontal aberrations commonly seen."

Dr. Beube's text is a welcome addition to periodontal literature and will do much to stimulate discussion among members of the profession.

A Manual of Oral Surgery. By W. Harry Archer, B.S., M.A., D.D.S., Philadelphia and London, W. B. Saunders Company, 1952, 643 pages with 1036 figures. $15.00.

The author has presented a clear, concise and practical manual of oral surgery.

The detailed illustrations of the various techniques are most graphic and instructive. They explain what would otherwise require lengthy and cumbersome descriptions.

Of particular interest are the well written case histories which illustrate and clarify the problems of diagnosis.

Dr. Archer's chapters on neo-plastic diseases, including both surgical and radiation therapy, are written by recognized experts in the various fields.

This text is informative and instructive and may be used to good advantage by both the general practitioner and oral surgeon.

MARVIN N. OKUN

A Manual of Dental Anesthesia. By W. Harry Archer, B.S., M.A., D.D.S. Philadelphia and London, W. B. Saunders Company, 1952. 192 pages with 146 figures. $5.50.

The author, a qualified teacher and clinician, has provided a practical manual of anesthesia for the dentist.

Dr. Archer's first chapter is a factual account of the history of anesthesia, a subject which has interested him for many years.

The first part of the book is devoted to local anesthesia. It includes a description of the variou anesthetic solutions, their use and post injection complications.

Part II, consisting of seven chapters, deals with general anesthesia and analgesia in dental surgery, both in office practice and in the hospital. Also included is information concerning patient preparation, complications resulting from the administration of general anesthesia and the technique for emergency tracheotomy.

An enlightening chapter on the legal aspects of anesthesia discusses the liability of the dentist or oral surgeon.

The text is recommended and should prove helpful to the dental profession.

MARVIN N. OKUN

Orthodontic Theory and Practice. By Samuel Hemley, D.D.S., F.A.C.D., Grune and Stratton, Inc., New York. 591 pages with 803 illustrations on 370 figures, and 13 tables. $14.50.

ORTHODONTIC THEORY AND PRACTICE is a new edition of Hemley's FUNDAMENTALS OF OCCLUSION, revised and considerably enlarged, which accounts for the change in the title. To the subjects discussed in the earlier work viz; normal occlusion, bone, development of the maxillae, forces of occlusion, etiology of malocclusion, classification, tissue changes and bio-mechanics, have been added bite-planes, the bite-plate, (an interesting distinction has been drawn between the two) and several chapters on the evolution of orthodontic appliances and orthodontic concepts.

The appliance per se no longer occupies a position of primacy usually accorded it in many text-books. "No special appliance is advocated because any appliance can be modified to serve advantageously if it is used in accordance with the biological limitations imposed by the reaction of the tissue on which it operates. The objective is the clarification of basic principles rather than the demonstration of finished technologies." A highly interesting and logical presentation of the evolution of orthodontic appliances shows that each new appliance has its origin in some previous inadequacy. Each appliance and each concept are described "in the language of its proponent. It is then carefully analyzed from the point of view of its accuracy, its validity and . . . its feasibility." Actually, these three criteria, accuracy, validity and feasibility become standard postulates which enable the student to arrive at rational conclusions. The critique on the concepts of the Tweed philosophy is so far-reaching as to constitute a challenge that can hardly be ignored in view of the overwhelming evidence that is offered.

The final chapter is a credo for the scientific (as contrasted with the empirical) orthodontist. It consists of fifteen statements on ". . . the hypotheses and concepts . . . substantiated by research in . . . biology . . . (to) determine the possibilities and limitations of orthodontic therapy." This credo can effectively chart a course which will avoid dogmatic empiricism on the one hand and pseudo-scientific persuasiveness on the other.

The lead editorial of the November 1953 issue of "The New York State Dental Journal" entitled "Inadequacies of some science texts," observes, "If we believe that oral and dental therapy is predominantly medical rather than a mechanical problem, our present approach to the study of a specialty of medicine is not entirely defensible." Such criticism certainly does not apply to Hemley's book which is rooted in the biological fundamentals. Since an orthodontic appliance is a device by which force is applied to move teeth, or which prevents undesirable tooth movements, anything that prevents or produces tooth movement is the concern of every dentist, be he specialist or general practitioner. This book, therefore, should prove of interest to every serious student in the field. It is indeed, a significant and provocative contribution to our dental literature.

DAVID TANCHESTER

Academy Proceedings

PHILADELPHIA SECTION HOSTS FOR 8th ANNUAL CONVENTION

The Philadelphia Section of The American Academy of Dental Medicine will be the hosts for the 8th Annual Convention to be held at The Penn Sherwood Hotel, in Philadelphia, from May 13th-16th, 1954. The theme of the convention will be "Diagnosis and Treatment Planning."

The Penn Sherwood Hotel is the former Penn Sheraton and has recently changed hands. The Convention Committee promises excellent food, attractive meeting rooms, and a 17-inch television set in each room.

A very interesting ladies' program has been arranged by the Convention Committee. In addition to the luncheons, dinners, and cocktail parties, the Committee has arranged a sightseeing tour of Philadelphia, luncheon at an historic inn, a tour of Valley Forge, and a matinee at The Hedgerow Theater.

The scientific sessions will be both interesting and enlightening. Each essayist on the program is a recognized authority in his field. All subjects will be discussed from a dental viewpoint.

Philadelphia has much to offer as a cultural, historic, and amusement center. Plan now to attend the 8th Annual Convention in the "City of Brotherly Love."

S. LEONARD ROSENTHAL, *Chairman*
GEORGE G. STEWART, *Co-chairman*

MEMBERS ELECTED AT MID-WINTER MEETING NEW YORK CITY, DECEMBER 6, 1953.

LUTHER H. BUTLER
Greensboro, N. C.

ARTHUR O. CHRISTIANSEN
Milwaukee, Wisconsin

HOWARD DIMOND
New Brunswick, N. J.

ARTHUR ELFENBAUM
Chicago, Illinois

JACOB B. FREEDLAND
Charlotte, N. C.

MORRIS GLICK
Brooklyn, N. Y.

ALBERT GOHO
Harrisburg, Pa.

FREDERICK T. HAMM
Detroit, Michigan

LEONARD M. HILLERSON
Philadelphia, Pa.

SAMUEL R. KLEIMAN
Chicago, Illinois

MARVIN MEISTER
Brooklyn, N. Y.

ROBERT A. NATHAN
New York, N. Y.

JEROME J. ORANGE
So. Orange, N. J.

ARTHUR RESNICK
Brooklyn, N. Y.

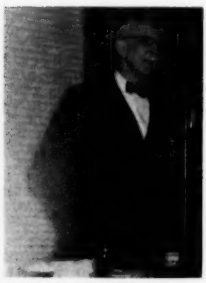
WILLIAM J. ROMANOS
Springfield, Mass.

JEAN SAYEGH
Philadelphia, Pa.

HOWARD F. SNEVEL
Cleveland, Ohio

SIGMUND M. WOLTER
New York, N. Y.

HIGHLIGHTS OF MID-WINTER MEETING—DEC. 6th, 1953



C. Ward Crampton, M.D.

Joseph I. Echikson, M.D.

Alonzo F. Myers, Ph.D.

Henry M. Goldman, D.M.D.

The American Academy of Dental Medicine's Mid-Winter Meeting and Luncheon held at the Hotel Statler in New York City on Sunday, December 6th, 1953 was an outstanding success. More than 200 members of the Academy and their friends heard an excellent panel discuss the problem of "Geriatrics."

The moderator of this panel was Dr. Joseph I. Echikson of Newark, N. J. Dr. Henry M. Goldman of Boston, Mass. spoke on "Geriatric Changes in the Oral Mucosa," while Professor Alonzo F. Myers of New York, N. Y. discussed "Planning for Retirement." The final speaker was Dr. C. Ward Crampton of New York, N. Y. who lectured on "De-Aging—What to do about age and aging."

MID-WINTER MEETING COMMITTEE

(Left to right, first row) Howard L. Ward, William H. Copperthwaite, Chairman; David Becker, Co-Chairman; and Anthony F. Posteraro.

(Second row) Sheldon J. Ross, George J. Witkin, Jacob A. Englander, Melvin J. Pomeranz, Abraham A. London, and Everett B. Simmons.

Announcements

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The Periodontia Department of New York University College of Dentistry which has been giving postgraduate courses in this subject since 1926, announces its full time course in Periodontia and Oral Medicine for one academic year, September 20, 1954 to May 26, 1955, leading to a certificate. Half-time two year courses are also given.

A three weeks full time course starting June 7th, 1954 is given; also a ten session (one evening weekly) introductory course begins February 24th, 1954. All courses are under the direct supervision of Dr. Samuel Charles Miller.

For information concerning the above or courses in other phases of dentistry write to Secretary, Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 10, N. Y.

INTERNATIONAL ACADEMY OF ANESTHESIOLOGY

The next meeting of the International Academy of Anesthesiology will take place on Wednesday, March 17th, 1954, at the Lloyd Dental Hospital, Seventh & Lloyd, Chester, Pennsylvania.

Information concerning membership in the International Academy of Anesthesiology can be obtained from the Executive Secretary, 355 East 149th Street, New York 55, N. Y.

DISTRICT OF COLUMBIA DENTAL SOCIETY

The District of Columbia Dental Society 22nd Annual Postgraduate Clinic will be held March 14-17, 1954 at the Shoreham Hotel, Washington, D. C.

Outstanding nationally known clinicians have been secured for this meeting and consist of the following: DR. LESTER W. BURKETT, Professor of Oral Medicine and Dean of the School, University of Pennsylvania School of Dentistry, whose subject will be "Oral Medicine"; DR. LEWIS ALEXANDER COHN, New York, N. Y., whose subject will be "Restorative Dentistry"; DR. IRVING R. HARDY, Professor of Prosthetic Dentistry, Tufts College, School of Dentistry, whose subject will be "Full and Partial Denture Prosthesis"; DR. LEWIS FOX, S. Norwalk, Conn., whose subject will be "Periodontia"; DR. GAYLORD J. JAMES, Cleveland, Ohio, whose subject will be "Practice Management"; and DR. DANIEL J. HOLLAND, Professor of Oral Surgery, Tufts College, School of Dentistry, whose subject will be "Oral Surgery".

In addition to these registered clinics, other attractions at the meeting will be table clinics, topic discussions, visual education, commercial exhibits, social entertainment, and an entirely new classification, research. Under this title, the latest dental developments will be shown and explained in detail by research men from the Federal Dental Services.

CHILDREN'S DENTAL HEALTH WEEK

During Children's Dental Health Week, February 1-6, 1954, there will be two Dental Health Expositions. One will be held at the headquarters of the Second District Dental Society at 111 Ft. Greene Place, Brooklyn. The other will be held at the Civil Defense Headquarters Building at 500 Park Avenue, corner 59th Street, in Manhattan.

The theme of the Expositions will be the "History and Development of Modern Dentistry". By means of newly created exhibits, motion pictures, lectures, demonstrations and actual bite-wing X-rays taken of children, the Children's Dental Health Week Committee will dramatize how dentistry can be made more comfortable for the patient. At the same time, all methods of prevention of dental caries will be stressed. This program will emphasize the four major points for improving the dental health of our children, (1) Toothbrushing, (2) Nutrition and Diet, (3) Fluorides and (4) Bite-wing X-rays and regular dental care.

TUFTS COLLEGE DENTAL SCHOOL

The courses in Periodontology listed below will be presented at Tufts College Dental School throughout the academic year of 1953-54. Application and further information may be obtained by writing the Director, Division of Graduate and Postgraduate Studies, Tufts College Dental School, 136 Harrison Ave., Boston 11, Mass.

DPG. 602—Participation Course in Periodontology
Monday through Friday, April 12-16, 1954
Tuition $150.00—Class limited to twelve
Dr. Irving Glickman and Staff

DPG. 603—Kirkland Gingivectomy Technique
Monday through Friday, May 17-21, 1954
Tuition $125.00—Class limited to twelve
Dr. Olin Kirkland

DPG. 604—Occlusal Equilibration
Each Monday for Five Weeks from Jan. 11,-Feb. 8, 1954
Tuition $150.00—Class limited to twelve
Dr. Edward T. Fischer

MARYLAND SECTION MEETING

A meeting of the Maryland Section of the American Academy of Dental Medicine will be held on Tuesday, March 16th, 1954, at 8:00 P.M., in the University of Maryland Dental School Building, Baltimore, Md.

Lt. S. Sigmund Stahl, U. S. Army, formerly of New York University College of Dentistry's Periodontia Department, will present a paper on "The Adaptation Syndrome and Its Relation to Oral Health." Included in this presentation will be a discussion of the use of cortisone for oral lesions.

All members of the Academy and their guests are cordially invited to attend.

In the Interest of

THE SCHOOL OF DENTISTRY
HEBREW UNIVERSITY, JERUSALEM, ISRAEL

A seminar is being presented on

"COMPLETE DENTURE PROSTHESIS FOR THE GENERAL PRACTITIONER"

Moderator: Raymond J. Nagle, D.M.D.
Dean of New York University College of Dentistry.
Diplomate, American Board of Prosthodontics.

Panel: Irving R. Hardy, D.M.D.
Professor of Prosthetic Dentistry, Tufts College School of Dentistry.
Diplomate, American Board of Prosthodontics.

Max Pleasure, D.D.S., M.S.
Chief of Prosthetics, Bronx Veterans Administration Hospital.
Associate Clinical Professor, College of Dental and Oral Surgery, Columbia University.
Diplomate, American Board of Prosthodontics.

James V. Giordano, D.D.S.
Member, New Jersey State Board of Dental Examiners.
Diplomate, American Board of Prosthodontics.

Questions will be answered from the floor

You are cordially invited to attend

Date: January, 14th, 1954
Time: 8:00 P.M.

Place: Henry Hudson Hotel (Grand Ballroom)
353 West 57th Street
New York City

FIFTH ANNUAL BERKSHIRE CONFERENCE IN PERIODONTOLOGY AND ORAL PATHOLOGY

The Division of Graduate and Postgraduate Studies of Tufts College Dental School will conduct the Fifth Annual Berkshire Conference in Periodontology and Oral Pathology at the Cape Codder Hotel, Falmouth by the Sea, Falmouth, Massachusetts, June 20-24, 1954. The Conference will consist of four days of lectures, seminars, and panel discussions regarding clinical problems by outstanding teachers and clinicians in dentistry, medicine, and their related fields. A special feature of the Conference will be a clinico-pathological seminar with microscopes provided for all participants.

The curriculum is arranged so that mornings and evenings are devoted to professional activities with afternoons open for recreation. This is a conference for general practitioners as well as those interested in a specialized practice.

For further information and application, write to:

The Fifth Berkshire Conference
Tufts College Dental School
136 Harrison Avenue
Boston 11, Massachusetts

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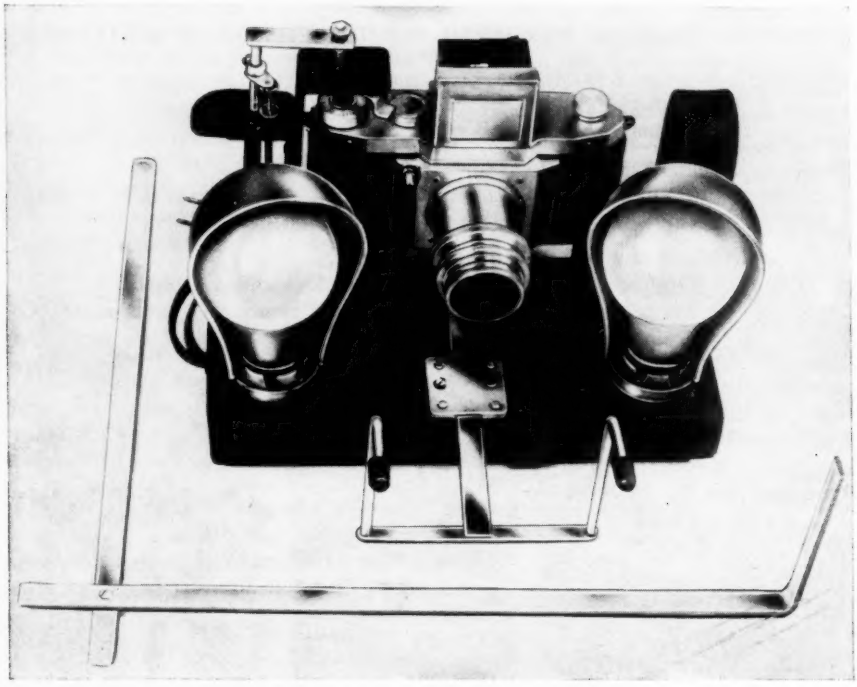

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